THE
LARYNGOSCOPE.

VOL. XLV OCTOBER, 1935. No. 10

THE TREATMENT OF OTOSCLEROTIC AND SIMILAR TYPES OF DEAFNESS BY THE LOCAL APPLICATION OF THYROXINE.*†

DR. ALBERT A. GRAY, London.

The purpose of the investigation was twofold. The first objective was to ascertain if, by producing an active congestion in the minute blood-vessels of the organ of hearing in cases of otosclerosis, any improvement in the hearing occurred, and thus test the correctness or incorrectness of the author's view of the etiology of the diseases, as expressed by him in the Dalby Memorial Lecture of May, 1934 (Gray: *Proc. Roy. Soc. Med.*, xxvii, 1934). The second objective, and from the human point of view the more important, was to find out if such improvement could be rendered of sufficiently long duration as to be of practical value in relieving these patients of their deafness in greater or less degree.

It should be added that the writer was encouraged to undertake this investigation by knowledge of the fact that cases of otosclerosis occurred in which striking improvement took place temporarily, under circumstances which brought, for the time being, an extra supply of blood to the organ of hearing. These were cases to which he gave the name otosclerosis paradoxica (Gray: *Jour. of Laryngol. and Otol.*, xxxviii, 141), and their

*From the Proceedings of the Royal Society of Medicine, Vol. XXVIII, pp. 1447-62. This report was made to the Section of Otology, May 3, 1935.

†This Investigation was carried out at the Ferens Institute, Middlesex Hospital.

occurrence offered a faint ray of hope that the condition might be produced artificially by some means or other.

For the purpose just mentioned it was first of all necessary to find some agent which, when applied locally, would produce an active congestion of the blood-vessels and at the same time be free from any irritating qualities which might cause inflammatory reaction. In this matter the writer was greatly helped by a clinical observation which he himself made when carrying out an investigation on quite another subject 30 years ago. At that time, being much annoyed by the symptoms of faintness and giddiness which frequently occurred when watery solutions of cocaine were applied to the mucous membrane of the nose, he made some experiments in order to find a more satisfactory way of applying the drug. After various substances had been tried, the difficulty was ultimately overcome by employing vaseline as the vehicle for applying the cocaine, and adding to the ointment powdered dried extract of the suprarenal glands. (Adrenaline at that time had not yet been isolated.) This ointment proved very satisfactory and the writer had no further difficulty with attacks of faintness and palpitation in his patients as a result of using cocaine. From the present point of view, however, the interest lies in a fact observed during the investigation just recorded. Among the substances which were tried in combination with cocaine, was dried extract of the thyroid gland. This substance, however, did not seem to have any effect in increasing the anesthesia, as did the suprarenal extract. But a different and unexpected result *did* occur. The patients noticed a slight increase in nasal obstruction which lasted for two or three weeks. On inspection of the nose the explanation of this obstruction was found in the fact that the turbinated bodies, and the mucous membrane generally, were in a condition of mild *active* congestion. That is to say, they were swollen to a moderate degree, such as is found in ordinary acute nasal catarrh, but the color was a bright red or pink, very different from the crimson appearance of a passive congestion.

The recollection of this clinical observation made by the writer 30 years ago proved invaluable in the present investigation. It indicated, without further search, an agent which apparently fulfilled at least some of the qualities necessary for the purpose. That is to say, it produced in the mucous membrane a prolonged active congestion and at the same time

caused no evident irritation. Obviously, therefore, it was natural to try the local effort of thyroid gland extract first, and, as the event ultimately proved, it was very satisfactory. In recent times, however, owing to the work of Kendall, Barger, and other biochemists, the active principle of the gland, thyroxine, has been isolated, and is now made synthetically, and this preparation is more satisfactory for practical use than the crude dried extract of the gland itself.

This explanation of the reason for employing thyroxine is necessary in order to prevent a misunderstanding. Otherwise it might have been assumed that the drug was applied for the purpose of compensating locally for a general deficiency of thyroxine in the patient's blood. Now, there is no evidence whatever that any deficiency of the natural hormone of the thyroid gland occurs in otosclerosis; and thyroxine was not selected in order to compensate for any such supposed deficiency. It was chosen solely on account of its local action as observed by the writer, and any other substance which has a similar local action would do equally well. But the writer does not know of any other such agent.

Thyroxine may be obtained from manufacturing chemists in the form of tabloids of 1-64 gr. (0.001 grm.) and 1-640 (0.0001 grm.), the first representing the official maximum dose. The writer usually employed half the maximum dose (1-128 gr.); and although this may appear somewhat large for individuals who are not deficient in their own natural thyroid secretions, it must be pointed out that they only receive the thyroxine once weekly. In any case no evidence of hyperthyroidism occurred in any of the patients, and indeed, most of them remarked how well they felt in general health as the result of the local treatment, and several put on weight as well. In one case a curious clinical fact is to be recorded (see Case 5). It was that of a married woman, a nullipara, age 25 years, in whom menstruation had always been very regular, and unaccompanied by any discomfort. When the treatment with thyroxine injections into the tympanic cavity was being carried out menstruation occurred every three weeks, and was accompanied by a certain amount of discomfort. Otherwise she felt remarkably well. The menstrual disturbance may have been merely a coincidence, or it may have been associated with the absorption of thyroxine, and further clinical observation on other cases will be necessary before a definite opinion can be

formed in regard to the matter. At present the clinical fact falls to be recorded. When it is pointed out that the patients only receive four or, at most, six injections, and that these are spread over a period of at least four or six weeks, it appears to be very unlikely that any serious harm could come from overdosage, unless a patient was already the subject of hyperthyroidism. So much does this appear to be the case that the writer intends in future to employ doses of 1-64 gr. in adults.

The method employed in the application of thyroxine to the ear consists of injecting the drug, suspended in 4 minims of distilled water, through the tympanic membrane into the inner middle ear by means of an ordinary well-made and smooth-working hypodermic syringe. The needle of the syringe must, of course, be a little longer than those used for ordinary hypodermic injections in order that the point may reach the membrane and project for the distance of 1 or 2 mm. into the middle ear. Furthermore, the needle must be bent at its proximal end in order that the line of vision be not obstructed by the barrel of the syringe or the hand of the operator. Another matter of some importance is the point of the needle. The bevel at the tip of the needle should not be long and tapering, but short and stumpy, somewhat similar to that of the needles used for intravenous injections. The calibre of the needle should be small. Needless to say, the proximal end of the needle must fit with air-tight closeness to the nozzle of the syringe.

The tabloid of thyroxine must be crushed down with a small spatula to the finest possible powder, otherwise some undissolved particle may block the needle at the critical moment when the fluid is being injected into the middle ear. The powdered tabloid, or as much of it as the operator wishes to use, is then mixed with not more than 4, or at most 5, minims of distilled water. If more than that amount of water is used, some is liable to escape down the Eustachian tube or back through the puncture into the meatus.

Anesthesia of the tympanic membrane is obtained by the instillation of 15 or 20 drops of the solution introduced by the present writer for this purpose many years ago. It consists of 10 parts of cocaine hydrochlorate dissolved in 90 parts of aniline. The aniline must be freshly prepared and should be colorless or of a very faint straw tint. And here a word of

caution is necessary. Aniline is a strong agent and, moreover, has a remarkable faculty of penetrating the skin. Indeed, it is this latter property which led the writer to employ it as a vehicle for cocaine for anesthetizing the tympanic membrane. Therefore, after the drops have been in the meatus, with the patient's head resting sideways on a table, for five minutes, it is very important to dry out carefully all the aniline and cocaine solution with cotton-wool mops. If this is done thoroughly, until no trace of aniline remains in the meatus, there need be no fear of symptoms of aniline poisoning. This is a point important to remember.

Before proceeding to make the injection, a broad cork is placed between the upper and lower jaws of the patient. The object of this is to keep the mouth wide open and thus prevent the patient from swallowing after the injection is made, which would permit a part of the solution to escape down the Eustachian tube. As wide a speculum as possible is then inserted into the meatus and the membrane well illuminated in the ordinary way by the forehead mirror. The point at which the puncture is made should be as nearly as possible in the middle of a line drawn horizontally backwards from the tip of the handle of the malleus to the margin of the tympanic ring. Thus, any possible injury to the long process of the incus or to the stapes is avoided, and at the same time the puncture is sufficiently above the floor of the tympanum to prevent any appreciable amount of the fluid escaping back through the puncture into the meatus.

In making the puncture it is important to remember that the plane in which the membrane lies is very oblique relative to the line of vision. Therefore, when the point of the needle is seen to be in contact with the membrane, it should be pushed backwards as well as inwards as the puncture is being made. This movement also allows the point to pass behind the bony lip of the round window and thus escape being impacted in the promontory. The fluid is then injected and a large proportion of it will lie in the space behind the round window and below the oval window. The needle is then withdrawn and the patient's head is immediately bent backwards so that he is looking up toward the ceiling. The reasons for this are to keep the fluid so far as possible bathing the region of the round window, and also to prevent it running down into the Eusta-

chian tube. The head is kept in this position for from five to 10 minutes, after which time the natural position may be resumed and cork removed from the mouth. If the procedure has been carried out successfully, the patient usually feels the presence of a little of the fluid in the nasopharynx and may even taste it in the mouth.

The whole procedure is painless, or as nearly so as possible. A slight feeling of giddiness is frequently present immediately after the injection, but this passes off in the course of a minute or two. Except for this there is no reaction and the patient can resume his work at once. In cold weather a plug of cotton-wool should be inserted into the meatus to prevent any possibility of the tympanic membrance becoming chilled; and the patient should be instructed not to blow his nose if possible for some time after injection.

In only one case was there any inflammatory reaction to the procedure, and in that case the trouble may, with justice, be ascribed, not to the injection of thyroxine but to the fact that within a few hours after the injection the patient put into the meatus 15 drops of radiostoleum that had been prescribed to be taken internally. There can be little doubt that the inflammation and suppuration which followed were the result of radiostoleum filtering through the puncture and were not due to the thyroxine. In proof of this is the fact that in this patient thyroxine had been injected on three previous occasions without causing any reaction. The inflammatory reaction was not very acute; the subsequent suppurative discharge dried up completely within two weeks, and the improvement produced by the previous injections of thyroxine gradually returned spontaneously.

Another contingency, which has occurred in two or three of the cases treated by the writer, is hemorrhage within the tympanic cavity. This is due to the point of the needle having punctured some minute blood-vessel either in the tympanic membrane or on the inner wall of the tympanic cavity. It is recognized by the deep crimson, or almost black, appearance which it gives to the lower portion of the membrane, associated with the fact that a pledget of cotton-wool applied to this region does not come out stained with blood, because the membrane is outside the clot. In none of the cases in which this occurred has there been any inflammatory reaction, nor

has it apparently interfered perceptibly with the beneficial effect on the hearing produced by the injection of the thyroxine. It is nevertheless, an undesirable incident, because, in the opinion of the writer, it would not be wise to repeat the injection until the clot was absorbed, for fear of the possible introduction of pyogenic organism with resulting middle ear inflammation. As the clot takes several weeks to disappear, the treatment must be correspondingly interrupted. It is doubtful if this hemorrhage can always be avoided, but the chance of its occurring is greatly diminished by employing a fine needle, by taking care that the point of the needle passes behind the bony lip of the round window and is kept very steady during the whole procedure.

When the meatus is narrow the procedure is obviously rendered more difficult. In none of the cases hitherto seen by the writer was the meatus so narrow that the operation could not be carried out, but in one or two of them it very nearly came into that category. It is inevitable, however, that sooner or later, cases will come under observation in which the field of operation is so obscured by narrowness of the meatus, that it would be unwise to attempt to make the injection blindly as would be the case in such a condition. Fortunately, such cases will be uncommon, and when they do occur special measures will have to be adopted to allow of the procedure being carried out.

Although inflammatory reaction does not occur after injection of thyroxine, there is sometimes a slight degree of headache on the side upon which the operation has been performed. This passes off in the course of a day or so.

It goes without saying that careful antiseptic precautions should be taken throughout. The meatus should be well mopped out with rectified spirit before the anesthetizing solution is instilled. Aniline itself is a powerful antiseptic and its faculty of penetrating deeply into the epidermis enables a thorough asepsis of the meatus and membrane to be obtained.

There is, of course, no satisfactory method of making the nose or nasopharynx really aseptic, and the most that can be done to avoid infection from this source is to be careful not to carry out the undertaking during the course of an acute nasal catarrh. Chronic nasal or nasopharyngeal catarrh apparently

does not tend to cause inflammation of the middle ear after the injection is made, and in several of the cases reported below the patients were suffering from this condition, and no inflammatory reaction occurred. The injections have usually been made at weekly intervals, so that each ear gets an injection once a fortnight. In some cases, however, the intervals have been a little longer, and this does seem to affect the efficacy of the treatment. If the hearing had not improved after an ear had had two injections, the treatment was discontinued.

Although the writer has given, in the preceding pages, a detailed description of the procedure, it must not be supposed that there is any difficulty about it. On the contrary the whole undertaking is simple in the extreme, and any otologist can perform it without any special training in the technique. The points to be observed are, first, to take care that the tip of the needle does not penetrate so deeply into the middle ear as to impinge upon the inner wall of the cavity, and second, to keep the syringe absolutely steady so that the tip of the needle does not wobble about. The piston of the syringe must run smoothly and easily in the barrel so that little pressure is required to inject the solution.

The writer carried out the treatment just described in 14 cases, and short reports on all these cases are given below:

Case 1: D. S., female, age 18 years, unmarried.

Aug. 1, 1934: Has noticed a gradually increasing deafness in the right ear for six months and in the left ear for one year. Occasional tinnitus occurs in the left ear, but not in the right. Paracusis has not been observed. Inflation and other methods of treatment have done no good.

Right Ear		Left Ear
Pale	Membrane	Pale
6 in.	Watch	2 in.
— 12 sec.	Rinne	— 18 in.
Lost below Sol -1	Low notes	Lost below Re 1
All heard well	High notes	All heard well
2 ft.	Whisper	1 ft.

Nov. 7: Patient thinks the hearing is getting worse.

Jan. 21, 1935: Injection of 1-64 gr. of thyroxine into left middle ear.

Jan. 31: Patient thinks the hearing is about the same as before. Injection of 1-128 gr. of thyroxine into right middle ear.

Feb. 14: Patient feels that the hearing in the left ear is better, and the fact is also noticed by her friends. Watch, 6 to 8 in. right, 3 in. left. Whisper, 3 ft. right, 2 ft. left. Injection of 1-128 gr. of thyroxine into right ear.

March 2: Tinnitus is now quite gone. Watch, 6 to 8 in. right ear, 4 in. left ear. Whisper, 5 ft. right ear, 2 ft. left ear.

March 12: Improvement maintained, but no further increase since last note. A blood-clot is present in the left middle ear.

Apr. 3: Watch, 1 ft. right ear, 4 in. left ear.

Case 2: S. F., male, age 29 years.

Sept. 19, 1934: Has been dull of hearing for 10 years, with very gradual onset. Had tinnitus in the earlier course of the deafness, but not now. Paracusis is only doubtfully present. Mother began to be deaf about the age of 60 years, but with this exception there is no deafness in the family. The right ear is rather the better hearing ear, but there is little difference between right and left.

Right Ear		Left Ear
Somewhat opaque	Membrane	Somewhat opaque
No rosy tint		No rosy tint
Perhaps a little indrawn		
Not heard by either air or bone	Watch	Not heard by either air or bone
6 to 8 in.	Whisper	6 to 9 in.
4 to 5 ft.	Conversational voice	4 ft.
— 22 sec.	Rinne	— 19 sec.
Slight loss	High notes	Slight loss but not quite so much as in right ear

Oct. 28, 1934: Injection of 1-128 gr. of thyroxine into left tympanic cavity. Some of the fluid escaped through the puncture back into the meatus. Patient felt a tickling sensation in nasopharynx a few minutes after injection.

Nov. 10: Hearing perhaps a little better in left ear. Conv. V. 5 to 6 ft. left.

Between Nov. 11, 1934, and Jan. 12, 1935, two injections of thyroxine were made, and the hearing cont'nued to improve.

Jan. 12 to March 2: Three injections of 1-128 gr. thyroxine made between these dates. Suppuration occurred in the left ear as the result of putting radiostoleum by mistake into the ear (which was ordered to be taken internally) a few hours after the injection of thyroxine had been made. The suppuration dried up within two weeks. In spite of this, the hearing, though not quite so good as noted in last entry, is very much better than it was originally and is again improving.

Apr. 27: Watch, right ear, heard only by bone-conduction, left, 1½ in. Whisper, 1 ft. right, 3 ft. left.

Case 3: Mrs. B. C. age 38 years.

Sept. 27, 1934: Dullness of hearing, of gradual onset, and increasing during the last 10 years. Tinnitus occurs frequently in both ears, but is not continuous. Paracusis is definitely present. There has never been any pain or discharge from the ears. There is no history of family deafness. The patient has frequently been treated by inflation of the middle ear per catheter but without any improvement. The right ear is the worse as regards hearing.

Right Ear		Left Ear
Possibly a little indrawn	Membrane	Possibly a little indrawn
1½ in.	Watch	3½ in.
— 25 sec.	Rinne	— 20 sec.
15 in.	Whispered voice	2 ft.

Oct. 3, 1934: Thyroxine (1-128 gr.) was injected into right tympanic cavity. No pain after injection, but a very slight feeling of giddiness which passed off in a few minutes.

Oct. 8: For a day after the injection the ear felt full, but there was no pain. Tinnitus began to get worse almost immediately after the injection. There was no giddiness or pain at any time. Patient's friends think she is hearing a little better, but patient herself is not sure of this. On examination a large blood-clot is seen through the membrane in the anterior part of the tympanum. Patient felt a certain amount of fullness in the left ear as well as in the right immediately after the injecion. Whisper, 2½ ft. right, 3 ft. left, so that improvement appears to have occurred in both ears.

Oct. 16: Patient is now quite sure that her hearing has improved, and her friends notice the same. The tinnitus is still present, but is less severe. No fullness in the ear. Watch, 4 in. right, 4 in. left.

Oct. 23: Hearing still improving. Watch, 5 in. right, 4 in. left. Whisper ca. 3 ft. right, ca. 3 ft. left. The blood-clot is still present in the tympanum, but is getting smaller. The patient is now greatly struck by the improvement in hearing, and she notices that not only does she hear voices better but other sounds also, such as knocking on the door, church bells, etc.

Case 4: E. S. male age 42 years.

Oct. 9, 1934: Has been deaf for 18 years. Deafness was of gradual onset, and began first in left ear. Never any pain or discharge from ear. Has been inflated frequently without benefit. No known family history of deafness. Tinnitus of a hissing character is always present. Paracusis Willisii definitely present, but not very strikingly so.

Right Ear		Left Ear
1½ in.	Watch	1 in.
4 ft.	Whisper	2 to 2½ ft.
— 6 sec.	Rinne	— 10 to 12 sec.
	Weber, to left	
Lost below Re (1#)	Low notes	Lost below Ut (2#)
Hardly any loss	High notes	Lost rather more than in right
Indrawn and rather opaque.	Membrane	Indrawn and opaque

Oct. 15, 1934: Injection of 1-128 gr. of thyroxine into left tympanic cavity.

Oct. 22: No pain after the injection. Patient has not noticed any difference either in the hearing or the tinnitus since the injection. Blood-clot within tympanum.

Nov. 12, 1934: No difference in either the hearing or the tinnitus. Blood-clot still present in the tympanum.

Jan. 28, 1935: Hearing and tinnitus remain the same as before. No further treatment advised.

Case 5: Mrs. K., age 25 years.

Oct. 16, 1934: Had been gradually getting dull of hearing during the last five years. No pain or discharge from ear at any time. Ears had been frequently inflated by catheter and the air passed freely up to tympanum, but produced no improvement. Paracusis Willisii strikingly present. Suffers from head noises which are worse when tired. Was operated on for some nasal trouble some years ago.

Right Ear		Left Ear
Normal	Membrane	Normal
2½ in.	Watch	2 in.
2 ft.	Whisper	1½ ft.
— 15 sec.	Rinne	— 18 sec.
Equal in both ears	Weber	Equal in both ears
Lost below fa (1)	Low notes	Lost below sol (1)
Very well heard	High notes	Very well heard

Oct. 30, 1934: Injection of 1-128 gr. of thyroxine into left ear. Slight feeling of giddiness for a minute or two after injection, and slight feeling of dullness and fullness in the ear, and a sensation of tickling in the throat.

Nov. 6: No pain in the ear after the injection or since, but very slight headache on the left side for a day or two. Patient and her friends say she is hearing better. Watch, right ear 2½ in., left, 2½ to 3 in. Whisper, 3 ft. left ear. Tinnitus about the same as before. No blood-clot inside the tympanum.

Dec. 12: Hearing distinctly better. Patient is now able to hear the wireless and such sounds as shutting of doors which formerly she could not hear.

Dec. 14: Improvement still increasing in left ear. Watch, 4 in. right, 2½ in. left. Patient notices that wax is now present in the ears, which formerly were dry. Injection of 1-128 gr. thyroxine into left middle ear.

Jan. 14, 1935: Whisper, 3½ ft. right, 4 ft. left. Injection of 1-128 gr. thyroxine into right middle ear.

Feb. 11: Watch, right ear 5 in., left 4 in. Whisper, right ear 3 ft., left 5 ft. The tinnitus is now very much diminished and hardly disturbs the patient at all. Injection of 1-256 gr. thyroxine into right ear.

March 12: Improvement maintained. Paracusis is not noticed and the tinnitus is almost gone. The patient is feeling very well and has put on weight. It is interesting to observe that menstruation, which formerly was very regular every four weeks and unaccompanied by any discomfort, now occurs every three weeks and is attended with a little discomfort.

Apr. 15: Improvement still maintained. Tinnitus remains absent. Watch, 6 in. right, 4 in. left. Whisper, 4 ft. right, 3½ ft. left. Low notes are now better heard: above si(-1) in right ear, and fa(1) in left ear. Wax is still being secreted in both ears.

Case 6: F. W. L., male, age 45 years.

Oct. 17, 1934: Has been deaf in the left ear since boyhood. Began to be deaf in the right ear about five years ago, and the deafness was accompanied by tinnitus. The latter is severe and is usually worse in the right ear, but occasionally worse in left. There is no pain. The tinnitus diminished under treatment with bromide, but the hearing did not improve. Paracusis is present. There is no deafness in the family. Inflation by catheter makes both hearing and tinnitus worse.

Right Ear		Left Ear
Opaque and indrawn	Membrane	Opaque and indrawn and an atrophic patch in ant. inf. quad.
Not heard by either air or bone	Watch	Not heard by either air or bone
Not heard at all	Whisper V	Not heard at all
1½ to 2 ft.	Con. V	6 in.
— 12 sec. ca.	Rinne	— 10 sec. ca.
Lost below mi(1)	Low notes	Lost below al(2)
Much loss above Mdh. 8.0 pft. 5.5 on Galton's whistle	High notes	Much loss Mdh. 8.0 pft. 7.0 on Galton's whistle
20 to 25 sec.	Absolute bone conduction	20 sec.

Oct. 31, 1934 to March 12, 1935: Three injections of 1-128 gr. thyroxine made at long intervals. On the whole, the condition was not improved, with regard either to the hearing or to the tinnitus. It is interesting to note, however, that occasionally the hearing would suddenly improve in a sriking way, and at the same time the tinnitus almost disappeared. These intervals, however, only lasted for a few hours, or a day at most, and were not very frequent. No further treatment, therefore, was advised. The case, in spite of the failure to give any lasting relief, is interesting on account of the intervals during which improvement occurred. It resembles in this respect the cases of otosclerosis paradoxica described by the writer (Gray, loc. cit.).

Case 7: V. W., female, age 31 years.

Nov. 14, 1934: Deafness began at the age of 18 years and tinnitus at 20. The onset was gradual and has steadily become worse. There has never been any pain or discharge. The general health is very good. Paracusis was present until a year ago, when an operation was performed upon the nose. Since then the paracusis has not been noticed, and the tinnitus has become worse.

Right Ear		Left Ear
Heard by bone only	Watch	Not heard by either air or bone
Rosy tint over promontory	Membrane	Rosy tint over promontory
4 in.	Whisper	4 in.
— 12 sec.	Rinne	— 15 sec.
Lost below fa(2#)	Low notes	Lost below si(2)
Great loss	High notes	Very great loss

Dec. 5, 1934: Injection of 1-128 gr. thyroxine into right middle ear.

Dec. 11: Hearing no better. Tinnitus perhaps slightly less. Injection of 1-128 gr. thyroxine in right middle ear.

Jan. 18, 1935: Hearing exactly the same as before treatment, and the same is true of the tinnitus. No further treatment advised.

Case 8: D. M., male, age 30 years.

Nov. 22, 1934: Deafness of very gradual onset began 12 years ago. Has had considerable amount of catarrh in nose and throat. Tinnitus not present. Paracusis not present; in fact, patient hears worse in a noise. Mother began to be deaf at the age of 30 years or before. Has one sister deaf, but she suffers from otorrhea. Has been inflated frequently, but the improvement lasts only a few minutes.

Right Ear		Left Ear
Normal or perhaps a little indrawn	Membrane	Normal or perhaps a little indrawn
1½ in.	Watch	Heard on contact only
9 in. to 1 ft.	Whisper	6 to 9 in.
22 sec.	Absolute bone conduction	20 to 22 sec.
—12 to 15 sec.	Rinne	— 12 sec.
Badly affected. Lost below sol(2)	Low notes	Badly affected. Lost below sol(2)
Well heard. None lost of Galton's whistle	High notes	Well heard. All notes of Galton's whistle heard

Nov. 26, 1934: Injection of 1-128 gr. thyroxine into left tympanum.

Dec. 6: Patient thinks he hears better and friends think so also. Watch 1 in. left. Whisper, 18 in. left. Watch 1½ in. right, and whisper, 1 ft. to 1½ ft. right. There is thus improvement for the whisper in the right ear as well as in the left. The improvement began on the second, third or fourth day, but patient is not quite sure which.

Dec. 8, 1934: Injection of 1-128 gr. of thyroxine into right ear. The improvement in the left ear is still present, but is stationary.

Jan. 12 to March 12, 1935: Two injections made into each ear during this period. Great improvement in the hearing has occurred. Wax, which was formerly absent from the meatus, is now being secreted freely. Watch, 3 in. right, 1½ in. left. Whisper, 18 in. right, 10 in. left.

Case 9: B. S., female, age 25 years, unmarried.

Nov. 27, 1934: Dullness of hearing first noticed in both ears, about three years ago. The onset was gradual. Paracusis is present, as also is distressing tinnitus. There is no family history of deafness. Inflation of the middle ear has been tried several times, but without any benefit.

Right Ear		Left Ear
Normal	Membrane	Normal
Not heard by air or bone	Watch	Not heard by air or bone
6 in.	Whisper	7 to 9 in.
15 sec.	Rinne	— 10 sec.
15 to 18 sec.	Absolute bone conduction	20 sec.
Lost below fa(1#)	Low notes	Lost below re(1)
Very slight loss	High notes	Very slight loss

Dec. 6, 1934: Injection of 1-128 gr. thyroxine into right ear.

Dec. 13: Had slight pain and giddiness on the day of the injection, but this had disappeared the following day. Patient noticed improvement in the hearing on the fourth day after injection. Watch, right ear ½ in. distance. Whisper, 8 in. Patient now hears from her house the traffic in the street which she could not do previously.

Dec. 18: The hearing is still improving and the tinnitus is much less. Her friends notice the great improvement in her hearing. Injected 1-128 gr. thyroxine into left ear.

Jan. 10, 1935: Improvement in hearing maintained and the tinnitus has almost entirely disappeared. Whisper, 9 in. right, 9 in. left.

Jan. 15: Injection of 1-128 gr. thyroxine into right ear.

March 12: Three injections have been made since the last note. Hearing is now greatly improved. Watch, 2 in. right, contact left. Tinnitus is almost gone.

Case 10: Miss T. B., age 15 years.

Dec. 5, 1934: Has always been a little dull of hearing, but the dullness has become worse during the last year. No pain and no giddiness. There is occasional tinnitus and paracusis Willisii is definitely present. There is one second cousin deaf, but otherwise no deafness is known in the family history. Patient had had the ears inflated frequently without benefit. The left ear is rather the worse of the two.

Right Ear		Left Ear
Rosy-pink tint on promontory	Membrane	Normal
9 sec.	Rinne	— 17 sec.
20 sec.	Absolute bone conduction	25 sec.
Lost below re(1)	Low notes	Lost below sol(1)
None on Galton's whistle lost	High notes	None on Galton's whistle lost
Heard only on contact	Watch	Heard only on contact
18 in.	Whisper	1 ft.

Dec. 10, 1934: Injection of 1-128 gr. of thyroxine into left middle ear.

Dec. 15: Some improvement in hearing. Watch, 1 in. from left ear; contact only in right. Injection of 1-128 gr. of thyroxine into right middle ear.

Jan. 9 to March 5, 1935: Five injections of 1-128 gr. thyroxine made. Hearing is now much improved. Watch, 1 in. right, 1½ in. left. Whisper, 3 ft. right, 2 ft. left. Patient notices that she hears the wireless much better.

Case 11: Mrs. W., age 31 years.

Dec. 11, 1934: Deafness of 15 years' duration. The onset was gradual, and at no time has there been any pain or discharge. Paracusis is definitely present but there is no tinnitus. Inflation of the middle ear has frequently been carried out, but without any lasting benefit. The hearing is worse when patient is tired. There is no deafness recorded in the family history. The watch is heard at a distance of 2½ in. in the right ear, and 1 in. in the left.

Jan. 5, 1935: Injection of 1-128 gr. of thyroxine into left middle ear. The patient, who is of a nervous temperament, fainted immediately after the injection. She admitted that this was due to nervousness and not to the pain, which was insignificant.

Feb. 14: No improvement in the hearing. It was decided not to continue treatment until the patient is stronger.

Case 12: S. S., male, age 13 years.

Jan. 14, 1935: Patient has been getting deaf very gradually for the last two years in the right ear, and recently the left ear appears to be becoming affected. Neither tinnitus nor paracusis has been noticed by the patient. There is no deafness in the family.

Right Ear		Left Ear
Slight rosy tint over promontory	Membrane	Slight rosy tint over promontory
6 in. ca.	Watch	9 in. ca.
+ 10 sec. ca.	Rinne	+ 12 sec. ca.
Fork heard better in right	Weber	Fork heard worse in left
Lost below si(-1)	Low notes	Lost below fa(-1)
Well heard	High notes	Well heard
27 sec.	Absolute bone conduction	35 sec. ca.
2 ft. ca.	Whisper	2 yd. ca.

Injection of 1-640 gr. thyroxine in right ear.

Jan. 18, 1935: Hearing remains the same as last note so far as the patient is aware.

March 6: Hearing remains the same so far as the patient is aware, but on examination he appears to hear the whisper now at a distance of 4 ft. from the right ear. Injection of 1-320 gr. thyroxine into left ear.

March 18: Letter from patient in which he says that he "thinks he hears a little better, but is not sure." No further treatment advised.

Case 13: G. M., male, age 21 years.

Jan. 15, 1935: Deafness began five years ago with gradual onset. Paracusis is present. Tinnitus was formerly present, but has now disappeared. There is no family history of deafness.

Right Ear		Left Ear
Normal	Membrane	Normal
2 to 3 in.	Watch	2 to 3 in.
15 in.	Whisper	12 in.
— 10 sec.	Rinne	— 12 sec.

The Eustachian tubes are clear. Inflation per catheter produces no improvement.

Feb. 2, 1935: Injection of 1-320 gr. of thyroxine into the left middle ear.

Feb. 13: No improvement of the hearing has occurred since last note.

Feb. 20: No improvement. The hearing is the same as before treatment. No further treatment advised.

Case 14: F. O., male, age 53 years.

Jan. 17, 1935: Has been deaf in the right ear since childhood, and in the left since about the age of 21 years. There is no known record of deafness in the family. Paracusis is present, but tinnitus is not.

Old dry perforation scarred over behind handle of hammer	Membrane	Normal
1 ft.	Conv. voice	4 ft.
Not heard	Whisper	2 in.
Much diminished	Bone conduction	Diminished

Blood-vessels along the handle of the hammer are not visible in either membrane. Injection of 1-128 gr. of thyroxine into left middle ear.

Jan. 21, 1935: Repeated injection of 1-128 gr. of thyroxine into left middle ear. Patient has occasionally had short intervals of improvement in the hearing last for an hour or two, but, except for these intervals, the hearing remains about the same.

Jan. 28: After the last injection fairly severe tinnitus occurred. The noise was of a "rushing" character and gradually passed off after a day or two.

Feb. 14: A little hemorrhge into the left meatus occurred after the last injection. As the improvement in hearing has only been intermittent as a result of the injections, it was decided not to continue treatment at present. This case is somewhat similar to Case 6. There were occasions when the hearing improved very much for a few hours. It therefore resembles the condition described by the writer as otosclerosis paradoxica.

Of the 14 cases above recorded, the hearing has noticeably improved in seven cases, and in the remaining seven cases no improvement has occurred. It is important to observe that in

those cases in which the deafness was accompanied by tinnitus, the latter symptom diminished or disappeared along with the improvement in regard to the deafness (see Cases 1, 3, 5 and 9).

Paracusis was definitely present in four of the cases in which the hearing improved. This fact is of practical importance, because that symptom is so well recognized by otologists as indicating a bad prognosis and little hope of any benefit from treatment. But in regard to the method of treatment described in this paper, this indication does not hold true, and those who wish to give the treatment a trial need not consider paracusis an unfavorable symptom when selecting the cases.

The return to functional activity of the ceruminous glands noted in Cases 5 and 8, is a very interesting clinical observation. The diminution of wax, with consequent dryness of the meatus, is usually, though perhaps not always, observed in otosclerosis; and when present is a characteristic feature of the disease. The fact of its return, therefore, is objective and conclusive evidence of the effect of the treatment on at least one portion of the organ of hearing. And it is difficult to see how the change could occur in any other way than by the return of a normal blood supply to the ceruminous glands.

An interesting feature of the cases which improved is the period of time which elapsed between the injection of the thyroxine and the first observation which the patient made of the increase in the hearing power. This period is usually of between four and eight days' duration. It is, of course, well known to pharmacologists that when thyroxine is administered by the mouth or injected into a vein, the drug does not begin to exert its effects until 24 hours have elapsed. After that time the effects gradually increase in intensity and reach the maximum at about the end of a week. The effects die away very slowly. This physiological peculiarity in the delayed action of the thyroxine when acting generally in the body is, therefore, in full agreement with the delayed action when applied locally, as in this investigation.

The age of the patient is a factor of considerable importance. The best results occurred in patients whose ages were between 18 and 30 or 35 years, although one very good result was obtained in a woman of 38 years (see Case 3). It is inter-

esting to note that the two patients in whose cases only intermittent improvement occurred were aged 45 and 53 years respectively (see Cases 6 and 14). But even among young adults, two or three did not respond to treatment.

Another interesting fact which this research has revealed is recorded in Cases 3 and 8. It will be observed that in those cases, when improvement occurred in the ear operated upon, improvement was also found in the opposite ear into which no injection had been made. When these patients said they were sure they were hearing better in the ear which had not been treated as well as in the ear which had, the writer was at first inclined to attribute it to imagination. But on testing the hearing carefully, it was found that the hearing in the unoperated ear was definitely improved. Furthermore, in one of these cases a sense of fullness occurred in the ear operated upon, for a day or so before the improvement was noticed, and a similar fullness was felt in the unoperated ear. On examining the literature of the subject the writer could find no reference to a similar phenomenon in recent times. But in his "Lehrbuch der Ohrenheilkunde," published in 1901, the late Professor Urbantschitsch, of Vienna, described a very similar condition. He observed that in some cases of otosclerosis—or dry catarrh of the middle ear as it was then called — the passage of the Eustachian bougie produced improvement in the hearing not only of the ear treated but of the opposite ear also. He considered that this was due to a reflex passing from one ear to the other, and termed it a crossed reflex. Little was known at that time of the functional vasomotor responses, and Urbantschitsch makes no reference to the nature of the reflex. In the light of the cases recorded in the present paper there can be little doubt that Urbantschitsch was correct in the observation of the facts. The subject, however, apparently did not arouse much interest in otological circles, probably because the cases in which the phenomenon was demonstrable were very few, and the condition itself was very transient.

Turning now to the other purpose for which this investigation was undertaken, that is, to provide a test for the writer's view of the etiology of otosclerosis, only a few words are necessary. The improvement in hearing which resulted from producing an active congestion of the parts provides such strong evidence in favor of that view that one may almost be justified in now considering it rather as an established fact

than as a hypothesis. The fact that many cases do not respond to the treatment does not appear to the writer to invalidate the hypothesis. The structural changes in the bony capsule of the labyrinth in the region of the oval window, and also those in the cochlear nerve, which result from the loss of the vasomotor response, may well have reached a stage at which repair is impossible. Furthermore, the vasomotor nerves themselves in these cases may have degenerated so far that the thyroxine fails to render them again functionally active.

For those who wish to try the method of treatment described in this paper, a few hints as to the selection of suitable cases may be helpful. It is not wise, for the present purpose, to attempt to distinguish cases of otosclerosis from the so-called chronic dry middle ear catarrh — if such a condition has any real pathological existence. When there is a noticeable loss of hearing for the lower notes, with retention for the high ones, associated with a negative Rinné, and with no serious diminution of absolute bone-conduction, along with absence, or only slight improvement, of the hearing on inflation of the middle ear, then the treatment by thyroxine injection may be tried. Cases in which the absolute bone-conduction is seriously diminished, even when the Rinné test is negative, are not likely to respond to treatment, and the same is true when there is any very noticeable loss of the hearing for high notes. The appearance of the tympanic membrane gives little help in the selection of suitable cases, and the writer has had good results when the membrane was quite normal in appearance as well as when it was thickened and opaque. It will naturally be understood that when the deafness has become extreme, no response to the treatment need be expected, for in those cases the structural changes both in the region of the oval window and in the nerve-elements of the organ of hearing have reached such an advanced stage that their capacity for functioning cannot be much affected by increased blood supply.

Another point which should be emphasized is in regard to the number of cases employed for investigation. Conclusions must not be drawn from the results obtained in one or two cases. The first two or three cases might all happen to fall into the category of successful responses to treatment or into that of failures, and undue optimism or pessimism, respectively, be established in the investigator's mind. When the writer began this research the first two cases happened to be

highly successful, but the optimism thus aroused was sobered by the following two or three cases which did not respond to treatment. It is necessary, therefore, to try the treatment on a fair number of cases before forming a judgment on the matter.

It is at present impossible to say how long the improvement will continue without treatment being renewed. In some of the cases reported the improvement has lasted several weeks and, at present, shows no sign of diminishing, and it is possible that it may be permanent. But in all probability two or three injections will have to be made throughout the year, because we know from the natural history of the disease that the deafness tends to progress slowly. It must be expected, therefore, that any treatment, however successful, would have to be renewed at intervals.

Summary: A large proportion (about 50 per cent) of cases of otosclerosis and so-called dry middle ear catarrh can be greatly improved in regard to both hearing and tinnitus by the intratympanic injection of thyroxine. Cases in which the disease is in its latest stages, however, do not respond to the treatment. The presence of paracusis Willisii is no contra-indication to treatment. The method of treatment is simple and can be carried out without difficulty by any otologist. It is practically painless or altogether so, and does not interfere with the patient's daily activities.

The rationale of the treatment depends upon the writer's view that otosclerosis is the result of a diminished blood-supply to the organ of hearing, consequent upon a gradual failure of the vasomotor responses. The action of thyroxine applied locally is to produce an active congestion without inflammatory reaction, and continuing for a long period of time.

It is not yet possible to say how often the treatment may have to be repeated. The improvement, when it occurs, lasts in some cases for several weeks, but sooner or later the effects must be expected to pass off. The present paper is of the nature of a preliminary communication, and the cases shown today will again be reported upon next session. In the meantime the writer hopes that others will investigate the matter and let the results of their experience be known.

Ferens Institute, Middlesex Hospital.

CLINICAL SYMPOSIUM.

1.—TWO CASES OF EARLY CARCINOMA OF THE LARYNX, AND A NUMBER OF CASES OF SO-CALLED ADENOMA OF THE BRONCHI, APPARENTLY CURED BY DIATHERMY.*

DR. JOHN DEVEREUX KERNAN, New York.

In connection with carcinomas in the larynx great emphasis is placed on early diagnosis. As a matter of fact, that is the case with carcinoma in any region of the body.

I wish to report two cases of laryngeal tumor as examples of early diagnosis of carcinoma. In addition, I wish to bring up a discussion as to what is best to do with such cases once the diagnosis is made.

The first case is that of a middle-aged man whose chief complaint was hoarseness, the duration of which was two months. Examination with the mirror showed a small tumor on the edge of the vocal cord, about its middle. This tumor was flat, sessile, and had no effect on the motion of the cord. The man had a positive Wassermann reaction. The tumor was removed by direct laryngoscopy, and the sections showed the condition revealed in the lantern slide which I shall show you. I am told by competent pathological authority that the irregularity in shape and size of the cells and their difference in staining qualities indicate a carcinoma. The question arose what should be done with this case. As the damage to the larynx was too little to call for a total laryngectomy, it was proposed to have the man come in to the hospital and to remove the cord by doing a laryngofissure. This proposal was refused by the patient, and he went to another hospital. There, examination showed no tumor, and a second biopsy showed no trace of the carcinoma.

The second case is that of a man, age 58 years, who had been hoarse for three years. He was a heavy smoker and used

*Read as part of a Symposium before the New York Academy of Medicine, Section on Otolaryngology, March 20, 1935.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 29, 1935.

his voice a great deal. I saw him at the beginning of this period when examination showed a uniform reddening and thickening of the vocal cord. He came to me again in January, 1934. Examination of the larynx then showed much the same condition, although there was perhaps greater thickening of the cords. He was under observation at short intervals until July, during which time the laryngeal condition had not changed. He was not seen again for two months. Then examination showed a very sinister development. There was a small, flat, sessile tumor on the anterior part of the left vocal cord. He was sent in to the hospital, and this tumor was removed by direct laryngoscopy. Although the biopsy showed it to be made up of a mass of epithelial cells, their size, shape, and staining qualities were not such that it could be said to be clearly a carcinoma. Again the question arose, what should be done? Should he have a cordectomy done through a laryngofissure? While we were considering this question, the cord healed up smoothly, and, at the present time, there is no trace of the tumor. Moreover, the enforced rest of the voice and the limitation of his smoking have resulted in a great improvement of the whole larynx.

In both of these cases, the whole little tumor and the surrounding tissue was removed. In fact, the whole vocal cord was stripped of mucous membrane. The question arises, should we be content with what has been done? Remembering MacKenty's report of a case under observation many years, which, in a short time after being pronounced normal, quite suddenly developed what eventually proved to be, in spite of a total laryngectomy, a fatal cancer of the larynx, one is naturally cautious in giving a prognosis. Should one do a cordectomy in spite of the favorable appearance, or should one be content to wait and observe? I hope there will be some discussion among the members of this question.

LUNG TUMORS.

The lung tumors which I am going to report have occasioned considerable controversy concerning their diagnosis and treatment. My material consists of 10 cases.

Microscopic Appearance: The microscopic appearance is characteristic of them all. They consist of anastomosing columns of cells which in places have a tendency to form gland-

like structures. These slides which I show you here illustrate the appearance very well. This first slide shows the anastomosing columns, as does this second one in which the columns are rather less distinctly marked. Here is a slide which tends to show well the formation of the glands. If I were to show you sections from any of the 10 cases on which I am reporting, they would appear very much like these. I think that a great deal depends on the angle at which the section is cut; some cuts showing the columns well, and others the gland formations. These tumors have been said by some pathologists to be carcinomas. On the strength of that diagnosis, I reported my first case as a carcinoma cured by diathermy and the insertion of radon seeds; other pathologists call them adenomas of a nonmalignant nature. It makes quite a difference as to what they really are, since the treatment proposed ranges from local removal to lobectomy or complete pneumonectomy.

Characteristic Place of Growth: They grow in the mucous membrane of the main bronchi from a broad pedicle, and their first spread is into the lumen of the bronchus. Only later, after they have gained considerable size, do they have a tendency to break through the bronchial wall and invade the lung tissue.

Their Manner of Growing: The slide which I show you now illustrates the manner of their growth. This one which you see is almost pedunculated; it is springing freely into the lumen of the bronchus and here at this point is growing outward between the ends of two cartilaginous rings. This second section has a much broader base and has clearly broken through the walls of the bronchus into the surrounding tissue. This third section also has a broad base, almost completely fills the lumen of the bronchus, but is still contained within it. It might be said here that these tumors never metastasize. Although they grow steadily for many years and may reach a very large size, it is always a local growth, never a metastatic one. I show you a slide here of a tumor which had existed in the lung of a woman for 36 years, she having been 42 years of age at the time of her death, and having had pulmonary symptoms from 6 years of age. The slides which I showed you before this one, and this slide, give the complete life history of these tumors. They show how they start as a localized growth in one of the main bronchi, break through the wall, and then continue to grow until they may

replace nearly a whole lobe of a lung. So much for their place of growth and manner.

Bronchoscopic Appearance: The bronchoscopic appearance is just as characteristic of these tumors as is their microscopic appearance and their place of origin. They are rather soft looking, moveable with a tube, and have a suggestion of pedicle about them. They are inclined to be vascular and bleed rather easily on contact with the tube. They never, as is the case with malignant tumors, cause a rigid fixed appearance on the bronchi. That is to say, they never do this except when they are very large, as was the case with the last tumor I showed you. As that is the only case that I have seen in which the growth reached such large size, I can not speak very authoritatively on this point, but only judge it must be so, as one can not imagine that so large a tumor would not cause considerable rigidity of the lung.

X-ray Appearance: The X-ray appearance varies greatly in these tumors, according to their situation and their size, whether or not they have as yet caused a blocking of the bronchus in which they are growing.

I am showing you here a slide of one seen in the right lower lobe of a girl, age 17 years. From the history we judge that it had been present for four years. You will see that it consists of a globular mass which has very little effect on the appearance of the rest of the lung. This girl was suffering from pulmonary hemorrhages and had had an exploratory bronchoscopy. The tumor mass was seen in the lower lobe and a specimen was taken. The report came back carcinoma of the lung. As we were much less well acquainted with these growths then than we are now, a poor prognosis was given and the girl sent home. She received half a dozen X-ray treatments, then nothing further was done. To our considerable surprise and delight, her pulmonary hemorrhages stopped, she regained her health, and at the present time, eight years later, is symptom free. Nevertheless, the tumor is still present in her lung, according to recent reports. This slide which I show you shows the condition four years after the last one. You will note that the mass is smaller and not quite so dense.

Very small tumors may produce a picture of great change in the lung, in fact one of much greater damage than may in reality exist. Here is a slide of the chest of a woman, age 35

years, taken in 1926. She gave a history of having had attacks of collapse of the left lung once or twice a year for five years. This picture was taken at the height of one of her attacks, when, as you will note, the lung is completely collapsed, showing almost no aeration at all. One would think from this picture that she must have a very large tumor. As a matter of fact, the tumor was comparatively small, but it was situated about the mouth of the left upper lobe bronchus in such a position that if at all swollen by any congestion, it would completely block the whole main bronchus. Evidently, her attacks, spread over the years, had been due to just such a blocking. This tumor was attacked with diathermy and radon seeds were inserted into its base. The result was eventually its complete destruction, with entire recovery of health. The woman is well at the present time, six years after the destruction of the growth.

This next slide shows the picture of the woman whom I have mentioned as having had a tumor for 36 years. You will see that the right lower lobe is completely solid, although the upper lobe is still aerated. Her chief symptom was hemorrhage. These became more and more severe, until eventually, she was completely bedridden.

This next slide is made from an X-ray of the same woman taken several years later and shows almost complete solidification of the right lung. The tumor in this woman was the size of a large grapefruit. The tumor in the last woman who also seemed to have a solid lung was the size of the end of one's little finger. This will bring to your mind very forcibly the fact that it is the situation, rather than the size of the tumor which may control the X-ray appearance.

Here is a picture which very strongly resembles that of the earlier condition of the last case. Her symptoms were those of chronic bronchitis, that is to say, cough, raising of sputum, and shortness of breath. You will note that the left lower lobe appears almost completely solidified. In this case, the tumor was comparatively small, but it filled the left lower lobe bronchus and produced the appearance that you see. After its destruction with diathermy through the bronchoscope, the lower lobe cleared to a great extent, as you may see in this picture. It is not completely clear, which was due to the fact that she continued to have a very severe bronchiectasis which

had been caused by the blocking of the bronchus and the consequent poor drainage. For a cure of the bronchiectasis, she ultimately acquired a lobectomy. This was successfully done, and she now has complete health. I think that it is of interest to call to your attention the fact that no trace of the neoplasm could be found in the lobe which was removed, showing that it had been completely destroyed by the treatment.

The next case which I call to your attention is very similar to this last one, except that there was a blocking on the right side, rather than on the left. Here, also, the treatment consisted of the insertion of radon seeds as well as the application of diathermy. The growth was completely destroyed, as was proved by a recent bronchoscopy. The lung shadow is not altogether cleared, due probably to the presence of the bronchiectasis. Fortunately, this is a dry bronchiectasis without symptoms.

To show you that this condition may exist, I am showing this slide, a picture of the lung of a little girl, age 13 years, who had one of these tumors in her left bronchus. Lipiodol has been injected and demonstrated the presence of a great many cavities in her lung. Yet, she is absolutely without symptoms, having neither cough, sputum, fever, nor shortness of breath. I think that it may be said that all of these cases have a bronchiectasis beyond the tumor. The question has been raised as to whether the tumor causes the bronchiectasis, or the irritation of the bronchiectasis causes the tumor. From the fact that several of my cases have been completely well once the tumor was removed, the conclusion may be drawn that it is the presence of the tumor that causes the bronchiectasis.

This next case shows the effect of partial blocking at the mouth of the right upper lobe. The symptoms consisting of cough and raising of sputum had existed for a comparatively short time. The tumor was found at the mouth of the right upper lobe bronchus. It probably blocked one of the branches leading to the lower part of that lobe. It was successfully destroyed in the course of a couple of months, and the patient is at the present time symptom free. The second picture shows the present condition of the lung.

Symptoms: In considering the symptoms of the tumors, it must be remembered that these tumors never metastasize and

that their symptoms are either hemorrhage or are due to obstruction of the bronchus in which they grow. These symptoms of obstruction may take two forms — the obstruction may be sudden and occur with all of the severity of an acute, massive collapse; or it may develop slowly and cause a bronchiectasis beyond the tumor. In the latter case, the symptoms then would be those of a bronchiectasis — the cough, raising of sputum, fever on occasions, and shortness of breath. Several of my patients have been treated in sanatoriums for tuberculosis.

Treatment: The question of treatment now comes up. I wish to remind you here that these cases on the strength of the microscopic appearance are often reported as carcinoma. Considering them as such, surgeons will propose pneumonectomy, and I believe several pneumonectomies have been done for these comparatively innocent growths. As a matter of fact, since they never metastasize, no such severe procedure is required unless they have been allowed to reach great size, or unless the bronchiectasis continues to cause severe symptoms, even after the removal of the tumor.

Some of these tumors have been successfully treated, simply by local removal with biting forceps. Jackson has reported such a case.

In my own experience, I have seen one disappear spontaneously. This was a tumor so large that it completely obstructed one bronchus and was beginning to fill the other. The diagnosis was confirmed by biopsy, the tumor being pronounced a carcinoma. Although this woman was very ill at the time the biopsy was taken, she was allowed to go home as any treatment was considered useless. She came back to the hospital about two years later, symptom free. A recent bronchoscopy has demonstrated the absence of any tumor formation in her bronchi. Such a case as this might make one think that no treatment at all is called for. As a matter of fact, however, unless something is done to relieve the patient of the presence of the tumor, it will eventually prove fatal.

I have treated the cases in the series which I have presented to you in several ways. As I have told you, one case disappeared spontaneously. One case had a short series of X-rays which relieved the pulmonary hemorrhages from which she was suffering; she has remained symptom free but

still has a tumor in her lungs. Two of the remaining seven cases had radon seeds inserted in the base of their tumors, as well as having diathermy applied. Both of these cases are well. I have used only diathermy on the other five, and, as far as can be told by bronchoscopy, the tumor has been completely destroyed.

It will be asked how can one know if the tumor has been destroyed. Since we know that they have a tendency to break through the wall of the bronchus and extend in the lung outside, it might very well be that the lesion in the bronchus would heal, the symptoms would cease, since there would be no obstruction in the lumen, and yet the tumor would continue to grow outside of the bronchus walls. If you will consider this slide which I show you now, you will see that in all probability, the electric energy which creates the heat in passing through the tissue from the large to the small applicator would concentrate both on the tumor outside as well as inside the lumen. That this has been so is proved by two cases in which it has been necessary to remove the lobe on account of persistent bronchiectasis. In both of these no tumor could be found, showing that it had been completely removed by the diathermy.

The diathermy is applied in this manner — the usual lead plate is placed on the outside of the body, usually at the chest wall, and, through a special bronchoscope which I show you, a long applicator is passed and applied to the growth. Owing to the arrangement of the bronchoscope and diathermy applicator, it is possible to keep the growth under observation all of the time that the current is on. When a proper appearance of blanching has been reached, either the procedure may be terminated, or the applicator moved to another part. I think that it is preferable to apply the diathermy to the whole growth without removing any part of it, as the presence of the tumor protects the wall of the bronchus from too great destruction. This occurred in one of my cases in which so much was destroyed that a stricture from scar occurred and caused the continuation of the bronchiectasis.

SUMMARY.

To sum up this series, there have been 10 cases: Two of these 10, following lobectomy, proved fatal, although the

tumor growth had been completely removed. Of the other eight, one recovered spontaneously, one was relieved of her symptoms by X-ray, and six were cured by diathermy, with radon seeds in addition in two of the cases. There has been a mortality of 20 per cent in the 10 cases, due, however, to lobectomy, rather than to the application of diathermy through the bronchoscope.

103 East 78th Street.

CASSELBERRY PRIZE FUND.

The sum of $500.00 having accrued from the Casselberry Fund for encouraging advancement in the art and science of laryngology and rhinology, said sum is now available, in part or as a whole, for a prize award, decoration, or the expense for original investigation or research in the domains mentioned above. Thesis or reports of work must be in the hands of the Secretary, Dr. James A. Babbitt, 1912 Spruce street, Philadelphia, before Feb. 1 of any given year.

CLINICAL SYMPOSIUM.

II. — A SERIES OF CASES OF TOTAL LARYNGECTOMY FOR CANCER.*

DR. ROBERT E. BUCKLEY, New York.

There are four possible methods of treating a carcinoma of the larynx.

1. Allowing it to take its normal course without medical or surgical interference, in which death is a certainty. This is obviously out of the question.

2. The treatment with radium, which I feel has proven of little value, although it has had a fairly good trial. If an occasional sporadic cure occurs it is the exception, and not the usual.

3. The treatment with deep X-ray therapy, in which the results as to permanency are still in doubt, but certainly some temporary cures seem to have been affected.

And lastly surgery, by which even to the most doubting, it must be apparent that hundreds of gratifying, complete and permanent cures are definitely on record.

Probably one of the largest series of laryngectomies, carried out by a single individual, was that brilliant series of the late John E. MacKenty, who was such a strong advocate of this procedure but only in well selected cases. I mention this fact because, unless the cases are well selected, the end-results in laryngectomy are relatively less satisfactory. Many years ago, the term intrinsic and extrinsic began to be used in describing a laryngeal cancer, and for the best statistical results it is well to remember that this operation is intended to be at its efficient point only when dealing with the intrinsic cancer. It is not my intention to mention numbers or percentages, as no two series could be parallel. Too many outside factors enter into the results, as for instance, the general

*Read as part of a Symposium before the New York Academy of Medicine, Section on Otolaryngology, March 20, 1935.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 29, 1935.

health of the patients in one series as against the health of another series, age against age, etc., and most important, the selection of the cases in one series as against the selection in the other. Dr. MacKenty had a strong tendency to be conservative, preferring not to operate if the case was at all extrinsic. This, combined with his marked ability as a technician, produced a series of extremely brilliant results, and even several years postoperative, the results show that cures are very numerous.

It is very difficult to lightly cast aside this fact, or the actual figures, which show so strongly the value of the operation of laryngectomy, until some equally efficient or more effective remedy is on hand to replace it.

In some recent years there has been a tendency on the part of some laryngologists to be more elastic in the type of cases chosen for operation, even though judgment shows that chances of a permanent cure are decidedly lessened by this elasticity. As time passes their statistics will not be as imposing as if they had adopted the more conservative attitude. I personally have probably been inclined to the less conservative tendency, as I have felt that in some of the definitely extrinsic cases, which happened to be radio-resistant, the possibility of at least prolonging life by surgery was still indicated, and in a review of these cases the procedure does seem to prolong the life of the patient, at least to a fair percentage. It would seem, therefore, that the results in general, over a long period of time, leave no doubt that a laryngectomy is an extremely valuable and efficacious operation, and I believe that up to the present time no other form of therapy will dare to claim results which compare in any way with this surgical procedure.

Judgment, however, has proven that all cancers of the larynx cannot be cured by surgery, and consequently it is necessary to divide these malignancies into two great classes: Those in which surgery is definitely indicated, and secondly, those in which surgery will probably prove useless. Two factors must be considered in determining in which class to place the growth: First, the location and extent of the mass is extremely important. Probably over 75 per cent of all cancers of the larynx occur on the true vocal cord, and so are at one period in their history intrinsic, and surgery is the indication; however, should the diagnosis be made at a later date, even

this intrinsic growth may have extended to the extrinsic parts of the larynx, and surgery will be of little use insofar as effecting a cure is concerned. A smaller percentage originate in the extrinsic portions of the larynx, and surgery is probably useless from the start. The microscopic characteristics must also be carefully studied in determining the probable outcome after laryngectomy, and it is found that a typical laryngeal cancer usually proves microscopically to be a squamous celled epithelioma, of varying degrees of malignancy, similar in every respect to a corresponding malignancy in any other part of the body. Thus, if we accept the classification of Dr. Broder, the tumor will be divided as the famous Wassermann test, into Types I, II, III and IV, depending on the degree of malignancy, which can be expressed numerically. In general, Types I and II, while they are malignant, they are not maliciously so; they have not made any great departure from normal tissue; they are a hard, resistant type, showing few mitotic figures, a large amount of connective tissue, or bridging, and, as mentioned above, are slow to give off metastatic bundles.

This is the type of growth usually found on the true vocal cord, and when so found, if it is still intrinsic, surgery is the definite, and I believe, the only indication, and the results obtained as to actual cures in any series of these cases will be extremely gratifying.

On the other hand, this type of tumor is extremely radioresistant, and treatment by deep X-ray is useless, since irradiation is after all only a selective third degree burn, the attempt being to burn up the tumor mass without burning up the surrounding neck tissue. This is obviously impossible in this class of tumor, as the neck tissue and the tumor mass too closely resemble each other to allow any selective burning of the one without a corresponding burning of the other.

In contradistinction to this last general class we have the soft tumors, classified as Types III and IV of the upper brackets of malignancy. This tumor is a soft, fast growing and extremely malignant mass, with numerous mitotic figures, only a small amount of bridging or connective tissue, and with a tendency to metastasize extremely early.

This tumor has definitely differentiated itself from normal tissue, and since it is usually softer than this normal tissue,

it is at least theoretically possible to consider its destruction by X-ray therapy. Clinically this is an extremely important fact, since these growths usually have their origin in the softer, extrinsic parts of the larynx, which, when added to its pathological characteristics, makes surgery undesirable.

The matter of certain types coming from certain locations of the larynx is clinically very interesting, and very important. If, as is usually accepted, the largest percentage of laryngeal cancers spring from the true vocal cord, it likewise holds that the largest percentage of laryngeal cancers must fit in the lower brackets of malignancy, and consequently it is obvious that in well over 50 per cent of all laryngeal growths, surgery would be absolutely indicated if any of these cases were seen reasonably early. Yet how pathetically few of these cases are seen early enough to offer them any surgical hope.

Many factors determine the ultimate results in a series of laryngectomies: A consideration and study of the patient's general condition, his age, heart and mental balance, with a particular attempt to have a reasonably normal metabolic quota. It is necessary to encourage the patient in every way as to the permanency of his cure, and so to place his mind in the proper, hopeful and confident mood. I am sure I have seen at least one case die several weeks postoperative because he did not want to get well.

In discussing his condition with the patient the use of the word tumor is decidedly more humane than the cold, hard word cancer, and should always be used, except with a very unusual individual. The use of mild drugs for several days preceding the operation always produces a desired effect.

In the actual performing of the operation of laryngectomy the technique is essentially the same in all cases, although numerous modifications are constantly appearing, the most valuable of which are the various improvements to lessen the amount of blood entering the trachea while operating, and the more advanced methods of draining the wound. One point that has been under discussion for many years, is the question of anesthesia, with its advocates on both sides.

Personally I have been more fortunate with my results with the use of the local anesthetic during the entire operation; however, it is just to say that, in the hands of experienced

operators, an immediate mortality is comparatively rare, regardless of which anesthesia is used.

In favor of the local anesthetic is the lessened possibility of a postoperative pneumonia; however, this is a comparatively rare complication. The argument against the local anesthetic is that the strain of the operation may produce a postoperative psychosis. In the series under consideration, two such psychoses developed, but in one case it followed a general, while in the other a local anesthetic, and in both cases the growth was so advanced that the duration of the operation was probably the actual cause of the complication. The patients practically always regain their normal mentality, although the derangement may exist for several weeks.

Thus passing over the operation itself, a consideration of the postoperative treatment is necessary, and this is probably more important than the operation itself. Experienced, hard working and intelligent nursing is an absolute requisite. The frequent use of catheter suction directly into the trachea, and the well regulated and frequent feeding by the nasal tube must be carried out by these nurses. A good diet, in a man of average weight, should contain a calorific value of about 2,000, with all the requirements of the body being present in their proper proportions. A small amount of water should be given before and after each feeding to prevent a mechanical plugging of the tube by the food. The diet sheet used at the Manhattan Eye, Ear and Throat Hospital contains all these requirements. Incidentally, it is practically the same diet used in any forced feeding, as for example in the swollen streptococcus sore throat, where mechanical interference demands the use of nasal feeding.

A complete blood chemistry must be done daily on the patient, seeking particularly an increase in blood sugar, which so rapidly will break down the surgical wound, to produce sloughing and gangrenous appearance. The not unusual appearance of increased blood sugar probably is produced by excessive thyroid disturbance, as the frequency of its occurrence was reduced to practically a minimum in a number of cases in which the isthmus of the thyroid was dislodged, and gently moved aside, instead of being ligated as is the usual procedure.

Most cancer cases occur in men past middle life, and so it is essential to guard against pneumonia, and for this reason

the patient must assume a sitting posture a very few days after the operation, unless there is some contraindication to this procedure. The treatment of the wound itself is the maintenance of constant and frequent dressings, and very adequate drainage.

Complications: Some of the complications were previously mentioned, such as psychosis, postoperative pneumonia, and increased blood sugar. To these must be added one which is now comparatively rare, namely, an infection of the mediastinum, which usually ends with a mortality. Its probable cause is lack of adequate drainage. Varying degrees of stomach and intestinal disorders may be counteracted by small doses of milk of magnesia, and a rearrangement of the diet, in an attempt to find a tolerance. In a certain small per cent of cases there is a marked drop in the respiration immediately postoperative, which must be watched for, and corrected by respiratory stimulants.

As can be seen from the above, excellent postoperative nursing and watching is essential.

CONCLUSIONS.

1. The operation called laryngectomy is no longer in its infancy, but is rather a well established and recognized procedure, with many hundreds of cancer cures standing to its credit.

2. To obtain the best results a preliminary biopsy is essential, not alone to definitely establish the diagnosis, but also to insure that very important microscopic study which is so necessary in deciding what therapeutic procedure shall be instituted.

3. A series of laryngectomies seems to indicate that surgery still definitely stands out as the most powerful foe opposing cancer of the larynx.

4. Although cancer is probably on the increase, it is a lamentable fact that there is a marked decrease in the number of patients who submit to laryngectomy, yet among these patients who refuse are bound to be a fair number of the so-called well selected cases in which a large per cent of cures would result. Their refusal is usually because they are unwill-

ing to permanently lose their voice, as is necessary following a laryngectomy.

Let us hope that in refusing this tried and proven operation these patients, now constituting the so-called selected series, do not pay a far greater price than the simple loss of their voice.

829 Park Avenue.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Cincinnati, Sept. 14, 1935. Fifty-seven candidates were examined, out of which number, 42 were certified and 15 conditioned.

The Board will hold an examination in Kansas City, Mo., May 9, 1936, during the meeting of the American Medical Association, and in New York City, October, 1936, just prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. Exact date has not been set as yet. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, Neb., for application blanks.

CLINICAL SYMPOSIUM.

III. — A SERIES OF CASES OF RADICAL MASTOIDECTOMY WITH SKIN GRAFT.*

DR. DANIEL S. CUNNING, New York.

Five years ago I had the privilege of reading before this Society a paper on skin grafting radical mastoid cavities. My enthusiasm at that time might have been interpreted as the ideas of a neophyte, but at the present time I feel that my convictions are even more strongly in favor of this procedure. The treatment of chronic suppurative mastoiditis is still a matter of extreme importance. Despite our ear clinics, school examination of childrens' ears, advanced bacterial knowledge, etc., the condition still remains extremely prevalent, so that in any of our larger ear clinics it is not unusual to encounter neglected, chronic, purulent ears with little or no hearing; the presence of cholesteatoma; acute exacerbations of chronic ears (polypi springing from the region of the promontory or oval window); recurrent polypi from any part of the tympanum, or facial paralysis occurring in a chronic middle ear suppuration. All of these foregoing signs or symptoms are, to my way of thinking, indications for a radical mastoidectomy. I have no doubt that in time the number of cases requiring radicals will be cut down immensely, due to a better knowledge of treating suppuration of the middle ear, but at the present time we see a great many ears that still require radical surgery.

Since most of the radicals occur in the working class, it is most important to reduce their period of hospitalization to a minimum, and return them to work as speedily as possible, owing to the present economic crisis. This matter of time is today an extremely important one, and I feel it is one of the main reasons for the use of the above-mentioned skin grafting operation. I venture to say that even in excellent surgical

*Read as part of a Symposium before the New York Academy of Medicine, Section on Otolaryngology, March 20, 1935.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication April 29, 1935.

hands a series of ungrafted radical mastoidectomies will still require an average of from two to four months before complete healing is established, and that during this period very frequent examinations and dressings, with the resultant inconveniences, are required. This is an entirely different picture from a similar number of cases, if the operation is supplemented by a skin graft.

For a great many years past, in every field of surgery, including otology, the transplantations of small islands of skin have been resorted to in an attempt to encourage the growth of epithelium. This has been practiced in radical mastoid cavities and, in a certain number of cases, was undoubtedly beneficial. In gross, however, the good accomplished has not seemed to warrant any marked enthusiasm. It was probably with this idea in mind that some 20 years ago Dr. Mosher, of Boston, first attempted to transplant a large block of skin, held closely in contact with the raw surface, in an endeavor to completely cover that area in one step. The value of his idea I feel has proven beneficial almost beyond words. His technique is described in detail in the *Transactions* of the American Otological Society of 1925 and THE LARYNGOSCOPE of July, 1931.

But, if I may give a brief resumé, it consists of the transplantation of the superficial layers of skin in a single piece, large enough to cover the mastoid cavity. This is done at the time of operation. A paraffin basket is moulded to the contour of the mastoid cavity; a suitable skin graft is then cut from the thigh, the mould is removed from the mastoid cavity, and the graft is gently laid over this wax impression with the external surface in contact with the wax. The impression, now covered with the skin transplant, is gently pressed back into the mastoid cavity; the external wound is closed in the usual manner. It is the intention of the operator to remove the paraffin mould through the meatus anywhere from the fifth to the tenth day.

It would seem pertinent at this point to mention a more recent method of skin grafting, brought out by Daggett and Bateman, of London, in which the graft is applied 14 days after the radical operation. According to their technique, the skin of the thigh is painted with 1 per cent methyline blue, and over this is spread a thin film of flexile collodion which is

allowed to dry. The methyline blue and collodion seems to offer an advance on our unsupplemented method of taking a graft, as the collodion stiffens the skin thus making it much easier to cut, while the methyline blue offers a distinct contrast to the pink and white color of the mastoid cavity, when the graft is being applied. The skin is stretched in the usual way and the graft cut. This procedure keeps the graft from rolling up at the edges and the deep and superficial surfaces can at once be distinguished. The graft is then placed in saline at 100° F. Obviously, this graft can now be cut into any shape or number of pieces desired by the operator, and it would seem that its main advantage consists in its ability to place each portion of skin wherever the operator desires to plant them.

For example, in an exposed facial it would at least be theoretically possible to injure the nerve with a primary graft. Daggett and Bateman's technique, therefore, would seem advantageous since it would allow the operator to place a small piece of graft above the nerve, another below it, another over the Eustachion tube, and so on until the cavity is practically covered with the exception of the exposed nerve. In this latter technique the graft is placed in position on the fourteenth day postoperative, via the external meatus, and is held in contact by the use of a melted wax, which is poured into the cavity following the implantation of the skin. This wax is made up of the following: B. naphthol, 0.25 gm.; oil Eucal., 2.00; olive oil, 5.00; paraffin qs., 100.

This melted wax sets quickly and holds the graft in place. Two weeks after grafting the wax is removed through the external meatus. It is of the utmost importance in using this technique to make a large meatus, as the posterior wound is closed at the time of operation. Since Daggett and Bateman close the wound, they feel that reopening it would automatically produce some oozing and bleeding, thus offering the possibility of a faulty taking of the graft. This bleeding is obviously eliminated by the application of the graft through the meatus.

As a matter of passing interest in two recent cases I have used a minor modification of the Daggett and Bateman method, namely, by placing the implant as suggested by them on the fourteenth postoperative day, but instead of closing the wound

at the time of operation it was allowed to remain open, and the plant was applied direct through the open wound instead of the meatus. In neither of these cases did oozing interfere with coaptation of the graft, and the results in both were very gratifying.

At the time of operation the posterior wound, as mentioned above, is left entirely open and is packed firmly with iodoform gauze which is allowed to remain for five days. Repacking again with iodoform is carried out every other day to the twelfth or fourteenth day. In the two cases in which this was done the wounds at the end of this time were clean and fresh looking, and healthy granulations were present over a fair portion of the cavity.

A comparative study of these different techniques seems to offer food for thought. For example, the primary graft as suggested by Dr. Mosher has two distinct advantages, namely, a single operation and consequently only one anesthetic, and, a saving of at least two weeks' time over the other methods. And this must be admitted to be a salient factor in the eyes of the patient. There is one dubious disadvantage, namely, the possibility of producing trouble by blocking off the drainage so soon after operation. Should we care to theorize it becomes apparent that all channels of drainage are occluded by the wax implant, thus offering the possibility of an intracranial complication. However, it is probable that the heat of the body melts the surface of the mould sufficiently to allow adequate drainage.

Another possible disadvantage of the primary graft is injury to an exposed facial nerve by pressure of the wax implant. The primary graft is therefore definitely contraindicated in the presence of an exposed facial, although there are certain cases in which this exposure, while present, is not visible to the operator.

Paradoxically, in a series of cases the writer investigated at the Manhattan Eye, Ear and Throat Hospital over a period of six years, no such complications occurred. And in a recent conversation with Dr. Mosher, while he still admits these complications as possibilities, he likewise says that so far as he remembers he does not recall one, although he and his confreres have been using this primary graft for the past 20

years. At this point it would seem necessary to state that a direct skin transplant to bare bone does take. I mention this because there is a false notion that the presence of newly formed granulation tissue is a requisite for the proper taking of a skin graft. In 72 cases investigated there was not one case in which the graft did not take in at least a fair per cent of its entirety.

Now to consider the secondary graft used by Daggett and Bateman. Its advantages lie chiefly in the fact that nature, having established her barriers of protection, the possibilities of any intracranial or facial nerve complications are reduced to a minimum, and this must be considered a very strong point in its favor. Likewise, since the graft is hardened and can be easily cut into any shape desired, it is possible to apply several pieces instead of a single unit. This is important where there is a visible exposure of the facial, in which case the graft may be applied above and below the exposure without coming in actual contact with it.

Its disadvantages are the waste of two weeks' time and the fact that it is not possible to apply a skin graft through the meatus as perfectly as through the posterior wound where every structure is in full view. Another disadvantage is the necessity for a second anesthetic as suggested by them.

In the third procedure in which the original wound is allowed to stay open for two weeks, and the transplant is then applied, two advantages would seem to suggest themselves. The first one is identical with the advantage attributed to the Daggett and Bateman method, that is, the probability of avoiding all intracranial and facial complications, and secondly the distinct advantage on the part of the operator of having an excellent view of the entire cavity, so that the graft may be applied exactly where he desires, and all structures are just as visible as at the time of operation and much more visible than if he were working through the meatus. Its disadvantages are two weeks of time required and the necessity for a second anesthetic.

In this series of 74 cases investigated at the Manhattan, 72 were grafted at the time of operation, and two were grafted by the external route two weeks after operation. It is interesting to note that there was not one case in which any untoward symptom or complication could be attributed to the skin graft,

although in this series at least twelve different men did the operating. One death, which was one of my own cases, resulted from a severe attack of erysipelas followed by a terminal pneumonia.

SUMMARY.

As a result of this series of cases I am firmly convinced that there is a definite use in radical mastoidectomies for the use of the skin graft, owing to the shortened duration of convalescence, the improvement in hearing in a number of our cases, a larger percentage of dry cavities, and lastly, the complications resulting from this procedure are extremely rare. As to which of these various methods will eventually be universally accepted, a further study and statistics in greater numbers will tell, although at the present time the writer feels that the secondary graft is safer.

145 East 54th Street.

CLINICAL SYMPOSIUM.

IV.—THE PRESENT STATUS OF RADICAL SINUS SURGERY.*

DR. E. ROSS FAULKNER, New York.

During the past few years the number of radical frontal sinus operations has steadily declined. This is equally true in private practice and hospital service. Fewer cases present the urgent symptoms which necessitate such an operation, and, no doubt, surgeons to some extent have become slightly more conservative in the matter of judging certain indications to be an imperative demand for radical surgery. The causes operating to produce this effect are many and one may not be able to estimate them all. It is probably more than a coincidence that with the advent of the general use of the ephedrine preparations during the acute stage of a sinusitis there has been a decrease in the more serious complications. The fact that syphilis is more certainly detected and better treated than formerly is also a factor of some importance. The wholesale removal of tonsils has also decreased the incidence of severe head infections and in general the more advanced information on food values has, no doubt, improved the nutrition of growing children and adults throughout the whole nation.

The cynical aspersions so often cast on radical sinus surgery, *viz.*, that the most of such operations were failures, I have never considered justified by facts, but have had some influence in the lay mind sufficient to prevent them readily consenting to an operation even when the most positive indications exist. In this they are often supported by the pseudo-conservatism of physicians who like to assume the role of being ultra-conservative in the matter of surgery; however, the radical sinus operation is by no means obsolete and it is incumbent upon every specialist to be able to take care of such cases when they come to him.

---

*Read as part of a Symposium before the New York Academy of Medicine, Section on Otolaryngology, March 20, 1935.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication April 29, 1935.

The choice of such operation on the frontal sinus will depend upon the pathology and anatomy of each individual case. The old Killian operation has given way somewhat to the more conservative Jansen or Lynch operation; but the latter is mostly suitable to cases of small or medium frontal sinuses. Where a very large frontal with necrosis of any portion of the anterior wall exists, the old Killian or obliteratory operation is the one of choice. The success of either depends on thorough eradication of all diseased tissue within the sinus cavity and especially the complete exenteration of all supraorbital ethmoid cells.

The Killian operation can be best performed by a double incision, the usual one through the lower part of the eyebrow supplemented by a long horizontal incision an inch above the supraorbital ridge and following one of the natural folds of the forehead. This leaves practically no scar if properly sewn up and gives one an excellent chance of cleaning out all the irregular recesses at the top of the sinus. Another advantage is the fact that you do not disturb the soft tissues over the bridge, hence, need have no fear of subsequent necrosis. It is possible by this method to leave a wide bridge and so have less deformity. The healing process should produce a complete obliteration of the frontal sinus cavity and is not likely to give any subsequent trouble. The nasal process should be removed in its upper part close to the septum to enable the soft orbital tissue to fill in the inner part of the cavity under the bridge to effect a complete obliteration with no space left communicating with the nose.

In the Jansen or Lynch operation, as you know, the floor of the frontal is removed and all the lining membrane removed from the whole cavity. This must be very thoroughly done and it is sometimes very difficult to be sure that all the crevices of the irregular area at the top are entirely cleaned out. The nasal process should be removed as little as possible as this prevents the intranasal opening from closing through the adhesion of the soft orbital tissue to the upper part of the septum. The method of healing in the latter operation is by the regeneration of a new frontal membrane while a good permanent opening into the nasal cavity should remain. In some cases with a very small frontal there may be obliteration of the cavity even after the Lynch operation and so effect the final result of the Killian. Where there is a regeneration of membrane a sub-

sequent infection may cause a swelling of the eyelid due to the absence of a bony wall on the floor to protect the soft tissues. This will very likely occur if the reinfection occurs a short time after the operation. Even an attack of hay fever may cause this phenomenon. Later on it is less likely to occur, probably due to some regeneration of the bony wall of the floor. If the intranasal opening does not become closed during one of these acute attacks the symptoms will clear up very quickly and will not necessitate any external drainage. Repeated reinfections after either form of radical operation may necessitate a second operation or a simple external operation with packing for a time can be tried. This may result in a cure, but is usually a long, tedious performance.

Let us now consider briefly the surgical procedures on the sphenoid sinuses. Here we have the same general fundamental condition to consider that prevails in the frontals except that there is no known surgical means of obliterating this cavity so the healing process must occur with regeneration of mucous membrane. The simple drainage operations by enlarging the natural openings downward and inward are most usually done. In such operations the lining membrane is not removed and the enlarged opening is taken care of during the healing process and not allowed to become too small for comfortable irrigation of the cavity. In cases with degenerated membrane the whole of the membrane must be removed as it would be in a frontal or an antrum, and the anterior wall removed as widely as possible. These cases will heal up satisfactorily with very little after care.

The cases of sphenoiditis which will give the most trouble are those with a definite osteitis. They are associated with the most pain and the lining membrane is thick and fibrous with a small amount of discharge. The opening in the anterior wall will persistently close up and require repeated reopenings. The lining membrane will not reform properly, but the cavity will continually fill up with infected granulations which reform as often as they are scraped out. Adhesions form between the floor of the cavity and the base of the sella turcica and leave a pocket in the posterior part with very poor drainage and persistent pain and symptoms of a focal infection. Repeated reopening of the cavity gives only temporary respite and an attempt at radical surgery becomes imperative. If both sides are involved, removal of the anterior half of the partition

between the two, with removal of as much of the floor as possible, is the only procedure which is likely to succeed. If only one side is involved removal of the floor is the best procedure.

The anatomy of some of these sphenoids is very badly adapted to surgery and some with extension of the cavity into the pterygoids or great wing of the sphenoid are almost impossible to cure completely. The very large unilateral sphenoid is another difficult type and seems much more prone to chronic infections than those with normal sized bilateral cavities. Removal of a portion of the floor will be indicated in many of these before satisfactory drainage can be established. In most of the sphenoid operations, unless the membrane has undergone complete polypoid change, it seems just as well to leave it alone and depend on drainage to restore the lining to a healthy condition.

There is much that is discouraging in the surgery of the sphenoids but I know of no surgical field where a more brilliant result may often be obtained not only in the relief of constant pain but in relieving the ill health attendant upon a focal infection in this region.

101 East 58th Street.

SEMON LECTURE.

This annual lecture will be delivered at 5 P. M., at the Royal Society of Medicine, London, on Thursday, Oct. 31, by Sir St. Clair Thomson. He has chosen for his subject "The Defences of the Air Passages."

A CASE OF SPONTANEOUS PERFORATION OF THE EARDRUM.

DR. PHILIPPE PANNETON, Montreal.

A few weeks ago I had the opportunity of seeing a case which to all appearances was one of spontaneous rupture of the eardrum.

Its very rarity would be such as to deserve publication. But it is even more important as it helps one in understanding the proper functioning of the tensor tympani; and every one knows how much work and study has gone in the investigation of the function of the intratympanic muscles.

Case: Miss G., age 30 years, telephone operator, was at her switchboard on March 12, 1935, when a dissastisfied patron "banged the receiver."

Miss G. felt an acute pain in the ear for one or two minutes, following which she had a sensation of "dead ear" for about half a minute. Pain came back then, accompanied by tinnitus, so that she had to quit work after an hour.

The ear was sore all night and kept her awake. However, there was no discharge of any kind. The day after, as the tinnitus was still there, as well as a slight pain, she went to a confrere who noticed what follows:

In line with the handle of the malleus one could see: *1.* A normal area 1 mm. wide, practically normal; *2.* A short line slightly darker than the rest, looking like a line of rupture. The line was curved following the direction of the circular fibres of the eardrum; *3.* Separated from the above-mentioned line of rupture by a very small area, appeared a red stain evidently an hemorrhage, representing an equilateral triangle pointing toward the umbo; the base of which was strictly parallel with the insertion of the membrane on the tympanic ring; *4.* A clear space between the base of the hemorrhagic triangle and the insertion of the membrane on its ring.

The patient came to me on April 1. At that time there could be noticed on the posteroinferior segment of the drum what

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 25, 1935.

looked like the scar of a tympanic rupture. Between this and the ring a red triangle which could be nothing but an hemorrhage gradually disappearing.

April 9: *Right Ear:* Eardrum normal. Very transparent and through which the incus is clearly to be seen. *Left Ear:* A few whitish lines. General coloring, normal. Where on April 1 appeared a scar, one could actually see a kind of blister. The hemorrhagic spot had lessened, but still retained its triangular shape. Low voice is hardly perceived at a distance of 1 m. with the left ear.

April 11: Very little change in appearance. Tinnitus that was very slight at the last examination is entirely gone. The only actual complaint is of a slight deafness.

TESTS WITH TUNING FORKS.

	R. Ear	L. Ear
Weber (435 v. d.)	←	→
Tuning Fork	Air — Normal	minus 12 sec.
	Bone — Normal	minus 10 sec.
Low Voice	Normal	1 meter

	Air	Bone	Air	Bone
16 v. d.	x	x	x	—
32	x	x	x	x
64	x	x	x	x
128	x	x	x	?
256	x	x	x	?
512	x	x	x	—
1024	x	x	x	—
2048	x	x	x	—
4096	x	x	x	—
XVII	x		x	

Barany test gives a normal answer. Equilibrium normal.

A thorough study of the case and of anatomy and physiology of the concerned organs led us to a somewhat bold diagnosis, but to one which appeared to be the only one possible. I put it down to a spontaneous rupture of the eardrum determined by a brutal reflex contraction of the tensor tympani caused by the sudden and excessive noise perceived.

Before suggesting an explanation, it would help to recall briefly a few anatomical and physiological facts.

Anatomy: The tympanic fibres are of two kinds: *1.* Radiating from the umbo towards the tympanic ring. This disposition gives to the central part more strength as the fibres

are denser near the umbo. *2.* Circular fibres. These fibres are slightly more sparse towards the center.

This layer, however, is much thicker near the bony ring so as to present a special formation called in French *bourrelet tendineux.*

The result of all this being that the maximum of resistance and thickness of the membrane is situated at both the periphery and center, while the minimum is approximately half way between.

We must add to this something peculiar, described by Gruber, special to the posteroinferior quadrant of the membrane, and which he named "dendritic formation." Almost always present, very strong, it reinforces the already strong distal region.

Physiology: Physiology of the tympanic membrane, however extraordinary it may seem, is not altogether clear. What part it takes in the transmission of sounds is not yet firmly established.

As for the ossicles, it is generally conceded today that their part is not to transmit the sound but to regulate the intralabyrinthic pressure through the fenestra ovalis.

Lately much has been done to help us understand the function of the tensor tympani muscles. One of Crowe's experiments is of unusual interest. It deserves a few words.

Using the principles established by Wever and Bray, in their so-called "Princeton Experiment," Crowe attached different weights to the tendon of the muscle and studied variations in the transmission of sounds from the exterior to the acoustic nerve of animals. He thus found that contraction of this muscle entailed an equivalent diminution in the sound transmitted.

A direct study of the tympanic reflex (aptly called malleolar reflex) was made by Luscher. The said reflex consisting in the drawing inwards of the whole membrane which is done by the malleus handle being pulled inwards by the tensor tympani muscle.

The reflex movement being set in motion by noise and appearing to be stronger when such noise is more violent.

All these make us understand easily what happened in the case of Miss G.

It seems evident that the cause could not be a direct trauma; a mere sound could not possibly determine the injury observed.

In cases of drum ruptures in caisson work, localization and appearance of the break or breaks seem to be different. Vail published two extremely interesting cases, accompanied with drawings which showed irregular perforations in the first, and linear ones in the other. But never before have we heard of a case giving the image of the one reported here.

Let us suppose that a violent noise determines a sudden contraction of the tensor tympani with accompanying movement of the malleus handle. Where will the maximal traction be felt? The malleus acting as a lever, the farther from the point of support, the more extensive the motion. Maximum will thus be at the umbo. Now where on the surface extending between the umbo and the tympanic ring do we meet the point of lesser resistance?

Exactly where we met the perforation in our case. Nearer to the umbo, the radiating fibres, more dense, give better resistance; the fibres themselves are stronger. Near the tympanic ring, the normal thickening is even assisted by Gruber's dendritic formation.

So that if there is to be a spontaneous rupture, the point where it is most likely to appear shall be midway between the ring and the umbo. Which happened.

As for the sequelae, we fear that the organ of Corti might have been damaged beyond repair.

Notre Dame Hospital.

MORTALITY OF MASTOIDITIS AND CEREBRAL COMPLICATIONS WITH REVIEW OF 3225 CASES OF MASTOIDITIS, WITH COMPLICATIONS.

DR. M. MARTYN KAFKA, Brooklyn.

The purpose of this paper is to bring before the medical profession the problem of mastoiditis and its numerous cerebral complications from various standpoints. There is no surgical disease which has so many varied opinions and views as regards the opportune time to operate. From my extensive studies of various cities in this country, New York State and some foreign cities, together with work done at hospitals, I find the mortality due to disease of the mastoid process is on the increase, in spite of the fact that rapid and revolutionizing advancement has been made in diagnosis and treatment of disease during the past decade.

The general practitioner fails as yet to realize that by waiting for a drum of an acute purulent otitis media infection to rupture he may be inviting some danger to his patient. The source of systemic infection of otitic origin in most cases can be found in the thrombotic process of dural sinuses that are first exposed to the infection that originates in the middle ear region. Through the transmission of infected thrombus particles or through repeated bacterial aggression, pathogenic organisms invade the circulation.[1]

Often we learn of doctors telling the patients that it is good to irrigate an ear which has not been opened. Pediatricians have been, more or less, very conservative about surgical intervention. From the Johns Hopkins Hospital I have received a report which extends over a period from 1915 to 1934. They have had 1057 cases of mastoids on their general service with 32 deaths. On the pediatric service during the same period they had 320 mastoids with 27 deaths. In other words, the mortality of the pediatric service almost equaled that of their

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 13, 1935.

general otologic service although the latter had almost three times the number of mastoid patients.[2] Yet G. M. Coates says that a bilateral mastoid operation should be performed by the otologist[3] on apparently normal ears on the simple demand of the pediatrician.

One otologist believes that intracranial complications are spread from the tympanic cavity and that the venous pathways of the temporal bone is significant as it is linked with spreading of infection. Early extension of involvement of the venous system can be recognized clinically and should serve as a milestone for early interference.

Abscess of brain may result from acute otitis media, though much less often than chronic otitis media, and it is hard to know just when the nervous system is involved.[5] Mastoiditis may result in osteomyelitis of the inner table of the skull with little production of epidural abscess and granulation tissue upon the dura. During generalized meningitis or brain abscess the nervous tissue connects freely with the subarachnoid space by means of perivascular spaces (Virchow-Robin spaces) during the period when pathologic changes are going on, and it is believed that along these channels of invaginated pia albuminous fluid is normally discharged into cerebrospinal fluid.[6] The infecting organism passes up this space about cortical veins to lodge, perhaps, deep beneath the surface and so the abscess may form superficially or deep in the white matter.[7]

The great problem of dealing with infants concerning the mastoid disease lies in the question of physical findings, as pain cannot with certainty (in infants) be localized. Pain on pressure is not reliable and, as well, is not a dependable and constant symptom in adults. A word of warning may be offered to those who feel that acute or chronic mastoiditis is to be considered lightly. It has been known for a long time that the course of many middle ear affections, meningeal processes, of specially light form, may appear that quite frequently disappear after a few days — sometimes after a few hours; although in some patients healing occurs only after some time. Alexander agrees with Wicart that meningitis may have a severe course, that power of resistance of the patient and his state of nutrition are of greatest importance and that in these cases chronic alcoholism blood, and constitutional

diseases (diabetes) will, without doubt, still more diminish the chances of recovery.[8]

Alexander gives his classification of meningitis with uncomplicated, acute purulent middle ear inflammation; also meningitis with uncomplicated, chronic purulent middle ear inflammation. The third type of meningitis with otic cerebral abscess, and the fourth type is meningitis with otic thrombophlebitis and extradural abscess. There is also a meningitis with labyrinth suppurations. In each of these groups three different types are found to be differentiated. Such types as retain anatomically visible connections of the ear region. There are types that do not show such connections and the tubercular forms of meningitis. Regarding the prognosis, one cannot expect to give at this time any information since two main factors must be considered, *viz.*, whether any micro-organisms are present, and their virulence; and whether by meningeal changes at time of operation can recognize a topical relation with the auditory organ. The form of purulent meningitis in which intradural infection starts from multiple inflammatory-plaques on pachymeningitis are unfavorable. In such cases are found in the ear region circumscribed or diffuse bone suppuration reaching the dura. The removal of these suppurative foci appears to afford no relief as was learned from an autopsy.[8]

Another otologist reports two fatal cases and believes that the combination of labyrinthitis and sinus thrombosis complicating suppurations of the middle ear occurs more frequently than is commonly believed. He studied seven cases of suppurative labyrinthitis histologically, and two present this combination. Labyrinthitis and sinus thrombosis may be independent of each other, each being caused by the original infection of the middle ear extending along different routes. When one is the different cause of the other, thrombosis of the sigmoid sinus is usually secondary to the labyrinthitis. He also feels that the reverse is true at times. He believes it is reasonable to assume that in some cases the development of intracranial complications secondary to labyrinth disease may be interdicted by proper treatment. But it also may be that if this intracranial complication is not secondary to the labyrinth, but merely is associated with it, and so cannot be stopped by any form of treatment aimed at the disease of the labyrinth. This seems to be one of the significant causes in

the high mortality rate in suppurative labyrinth, especially in cases of acute nature. He feels that the most commonly chosen paths of infection described by him are preformed pathways (ductus and saccus endolymphaticus as well as the labyrinthine veins), and the newly formed portals of infection-erosion of the bony capsule of the labyrinth by fistula from within outward, through the semicircular canal, usually posterior and through the body of the labyrinth.[9]

From those cases I have studied at New York University and Bellevue Hospital Otological Service, there were admitted over a period of 21 years (from 1912 to 1933 inclusive), 3225 patients suffering from both acute and chronic mastoiditis; these number of cases taken out of a total number of 1,060,000 hospital patients admitted over a corresponding period. Out of this number 2100 were of acute mastoid inflammation, and 1125 were of a chronic nature. Each one of these patients were carefully observed and treated at the hospital with studies being made ranging with X-ray examinations, blood cultures, blood counts, Wassermann tests, urinalysis, and various consultations were conducted when indicated, and operations were performed in almost all of the cases. From the total number of 2100 primary acute mastoiditis, there were 126 cerebral complications, 90 of which were fatal thus:[10]

Brain abscesses, 16; cerebral hemorrhage, 1; cerebrospinal meningitis, 49; lateral sinus thrombosis, 14; sigmoid sinus thrombosis, 1; cavernous sinus thrombosis, 1; chronic mastoiditis, 1; septicemia, 6; cerebral thrombosis, 1. Total mortalities, 90.

There were 36 patients who recovered of this group of 126 cerebral complications which were as follows:

Brain abscesses, 2; cerebrospinal meningitis, 6; lateral sinus thrombosis, 22; chronic mastoiditis, 4; septicemia, 1; cerebral thrombosis, 1. Total recoveries, 36.

In other words, of a total of 2100 primary acute mastoiditis cases, about 16.66 per cent developed cerebral complications; and out of the 126 complications there were 28.55 per cent recoveries and 71.45 per cent mortalities.

There were 1125 chronic mastoiditis cases admitted to Bellevue Hospital in 21 years, out of which there were a total of

83 cerebral complications developed, and out of this number 66 were fatal and 17 patients recovered thus:

Complications	Fatal Cases	Recoveries
Brain abscesses	11	4
Cerebrospinal meningitis	15	—
Cerebral meningitis	17	4
Pachymeningitis	2	1
Lateral sinus thrombosis	5	4
Septicemias	3	—
Cerebral thrombosis	2	2
Labyrinthitis	1	2
Total	66	17

Out of the total number of the chronic mastoiditis admitted 13.55 per cent developed cerebral complications; and of the 83 complications in this series 75 per cent died and 25 per cent recovered.

The term brain abscess should be strictly confined to a collection of pus within the brain substance, but for practical purposes any such process within the cranial cavity is so considered. Brain abscess may be extradural, subdural, and intercerebral or cerebellar.[11] A great number of these abscesses may arise by extension in various ways from chronic otitis media. Abscess of the brain may occupy any region, but according to Kanavel,[12] 60 per cent are in the temporal lobes, 25 per cent in the cerebellum and the balance, 15 per cent, in frontal lobes and elsewhere in the brain. The infective organisms are usually ordinary pus cocci, sometimes, the pneumococcus; rarely the colon bacillus which may give rise to abscess cavity filled partially with gas.

The prognosis of the abscess within the brain, (intracerebral) (or intracerebellar), is serious; the mortality statistics of recent years averaging well over 50 per cent, although about 43 years ago, Sir Wm. Macewan[13] submitted a much more favorable result. Eagleton,[14] in 1922 reported 75 per cent mortality in brain abscess patients; while Dench[14] in May, 1929, gave a report of 21 patients suffering from cerebral abscess with nine recoveries of this series. Recently Cahill also reported 12 patients who suffered from brain abscess of cerebral and cerebellar types, and he attributes Mosher's wire gauze cone as a drain, as being the cause for the cures. Even as early as 1893, Macewan,[13] reported 19 cases of cerebral and cerebellar abscesses, with only one mortality of the entire

series. As yet, up to this advanced stage this exceptional record has not been improved.

The causes of death from brain abscess with or without operation are as stated by von Bergman: Rupture on to the surface of the brain with resultant meningitis; rupture of abscess into the lateral ventricle. Failure to find the abscess at operation. Occurrence of secondary abscess. Existence of more than one chamber in the abscess. Sinus thrombosis with pyemia. Pneumonia. Shock. Respiratory failure. Most[15] favorable prognosis is in patients whose abscess is near surface of the brain, especially when a fistula through the dura is present, and where a firm capsule about the abscess (which is really an abscess with a stalk), is present. Unfavorable abscesses are those which are deep into the brain substance, and where no capsule is present.

Leptomeningitis,[16] or meningitis due to extension from otitis media, and mastoiditis, tends to be more localized than the hematogenous variety; and the organism generally is staphylococcus or streptococcus, rarely the pneumococcus.

C. C. Coleman[14] believes that the drainage of brain abscess should be accomplished by direct approach, with the least possible trauma to the surrounding brain tissues. This, he feels, will assist in decreasing somewhat the mortality due to brain abscess. He thinks where the drainage is accomplished with great difficulty, tapping of the abscess, as proposed by Dandy, with a later insertion of the tube, is to be preferred. In recent years, too much emphasis has been placed upon the type of drain and too little concern attended with the avoidance of trauma in the drainage of the brain abscess. In the recent surveys, surgical literature points to simplicity in treatment of this condition; and in the end the simple methods are really the best for obtaining good results. Experience teaches that it is best to await the formation of the capsule in the primary stage before attempting an operation. An earlier venture, before the capsule has formed, spells, as a general rule, fatality; however, Eagleton,[17] feels that it is important to have a more exact knowledge of routes of extension from the middle ears; for it will reduce exploratory punctures to a minimum.

Meningitis still remains an existing menace, while there are circumstances when an abscess may be well drained but the patient dies and postmortem examination discloses there are

multiple infections elsewhere in the brain. There[18] are instances when a patient may leave a hospital in a seemingly cured condition but may return several months afterwards or a year later showing signs that the abscess had refilled due to original drainage not being continued over a longer period in the first place.

It is often difficult to differentiate between an otitic purulent meningitis, tubercular meningitis with suppurative otitis. In tubercular meningitis symptoms are less acute, and there is a more likely involvement of the cranial nerves at the base with pressure symptoms more common. Tubercular meningitis resembles brain abscess with low temperature and slow pulse. The cerebrospinal fluid is usually under pressure. It is perfectly clear or slightly opalescent. The[19] cells in the spinal fluid are almost all lymphocytes. In purulent meningitis cells are mostly polymorphonuclear. Otitic meningitis is differentiated from meningococcus meningitis by the presence of meningococci in the cerebrospinal fluid. In a brain abscess the temperature is usually very low compared to the other conditions, and the pulse is slow with rigidity of the neck. Otitic meningitis is more virulent and runs a more rapid course, and the type of meningitis complicating acute otitis, as a general rule, takes a shorter course.

Meningitis complicating chronic otitis might be fatal within 48 hours; few last longer than 10 days to two weeks. Cases occur in which there are no meningitic signs, and the patient feels well, then suddenly becomes unconscious; shows signs of advanced meningitis, and dies in a few hours. This I will attempt to prove in actual occurrences in a series of the cases which I am reviewing at Bellevue Hospital Otological Service. Almost all cases of meningitis following acute otitis media die. This I have already shown in this paper, namely that out of 55 patients suffering from cerebrospinal meningitis, complicating acute otitis media, 49 were fatal and six patients recovered. Out of 32 meningitis complicating chronic otitis, only four recovered.

Brieger[19] collected 71 cases of patients who had otitic meningitis, and found six were cured and one of this group of six complicated acute otitis, and five of this group followed chronic otitis media. The labyrinth was involved in four of the total cases. There was a temperosphenoidal abscess in two.

Koerner[19] found 69 cases of temperosphenoidal lobe abscess to 30 of cerebellar abscess. Gower found 186 temperosphenoidal lobe abscess to 41 in the cerebellum.

Sinus thrombosis occurs more frequently in cerebellar abscess, 10 times in 59 cases, (Hessler).[20] Oppenhein, 1897, stated that brain abscess is far from an invariable accompaniment of sinus thrombosis occurring only in five patients out of 34 cases of thrombophlebitis, (Jansen).[20]

In the formation of thrombus, the inflammations of the vessel wall, especially that of the intima, play important parts. The dura of the vessel wall is only thick, so that infectious material when it remains for any period of time in contact, the penetration is facilitated. There is a septic process brought about by the fine vessel system in the septa of the pneumatic cells; or of those which empty into the sinus before sufficient time for formation of macroscopically noticeable thrombus had elapsed. These[21] minute vessels get diseased when passing through bone in the process of purulent disintegration; the outcome will be phlebitis and periphlebitis.

Hays[22] in his paper emphasizes that even when a simple mastoid operation is performed with no exposure of the sinus, an infection of the vein can take place. When an original wound has healed as late as six weeks after operation, a blood culture may show a few organisms. Indefinite symptoms may arise which later may prove to be of serious consequences. In patients where signs of vein infection arises some few weeks following operation, the complications might be more serious than the ordinary types of thrombosis. Consentrating on the vein does not always ward off complications. Where transfusions are used, it assists the blood to throw out bacteria from the blood stream but it may fail to stop development of sequelae.

Grant[23] in his report based on a series of 51 cases, verified abscesses of brain, states that (in 39 cases of neurosurgical service of the University Hospital, eight from the Graduate Hospital and four from Mt. Sinai Hospital), of this group only 39 patients' diagnosis were preoperatively confirmed; in four patients the lesion was considered a tumor. All 43 patients suffering were operated on, and abscesses drained. In the balance of this series of eight cases, one child was admitted unconscious, with signs of increased intracranial pressure.

History obtained was incomplete; a ventricular tap was performed for relief of pressure, so that better means and more time could be obtained for proper study of these types of cases; but the child died six hours later, and the postmortem examination showed a cerebellar abscess, yet the author could not find in this paper any mention of otitic origin concerning this case. In the other two cases of the remaining eight, a diagnosis of meningitis was made on admission and no operation seemed indicated; but autopsies demonstrated a subcortical abscess of the frontal lobe, rupture into the anterior horn of the ventricle and a subdural frontal abscess with generalized meningitis. The last of this series was a cerebellar abscess patient who died suddenly, while awaiting operation, from cerebellar crisis.

The pathway of infection to the brain to the temperosphenoidal lobe usually is through the roof of the middle ear or from the roof of the mastoid antrum.[24] Temperosphenoidal lobe abscess is either an extension from epidural abscess in this location or the result of an extension through the vessels which pass through the roof of the middle ear or mastoid antrum. Infection from the middle ear and mastoid may extend to the cerebellum through the internal ear, lateral sinus, the inner wall of the mastoid antrum, or via an epidural abscess or through the facial canal. Fifty per cent of cerebellar abscess complicating chronic otitis are extensions from the labyrinth. Of acute otitis complications related to cerebellar abscess there are about 5 to 10 per cent; the balance takes origin from lateral sinus thrombosis or epidural abscess of posterior fossa. Of 19 cases of cerebellar abscess following acute otitis, Neumann found that eight were due to sinus thrombosis and six followed epidural abscess in the posterior fossa. When sinus thrombosis causes cerebellar abscess, the infection may spread from the interior of the sinus through its posterior wall to the cerebellum; or the thrombus may extend from the sinus to one of the pial veins and infect the cerebellum through this vessel.

The writer has seen a number of cases at Bellevue Hospital which were, indeed, very unusual and interesting concerning mastoiditis following fracture of the skull.

I have attended four cases during the past year which terminated in acute mastoiditis and a history of direct trauma

was obtained from the patients. One case, a man, age 50 years, laborer, had been struck by an automobile which was going about 25 miles an hour, (so stated the patient), came under my care with symptoms of bleeding from his right ear, and pain on the right side of his head at about the temporal region. He was admitted to the hospital and treated for fracture of the skull. About three weeks later he developed acute mastoiditis on the right side, with signs of acute labyrinthitis. He was operated on for acute traumatic mastoiditis and recovered without any untoward symptoms. The labyrinth was not done, but expectant treatment was followed. Another patient, age 29 years, was studied. He recovered, following right acute mastoiditis from a traumatic condition to his head which occurred after an automobile accident. He developed pain and tenderness over the right mastoid region with a serosanguineous discharge from right canal associated with marked pain upon slightest palpation over the right mastoid and temporal region. His right canal was filled with blood and serum. A right modified mastoidectomy was performed 21 days after he was admitted to the hospital; also a ligation of the internal jugular vein. Spinal fluid was tested and Wassermann was negative. After 51 days the patient recovered and was discharged. Another patient, male, age 36 years, recovered from a left acute mastoiditis following trauma. He fell down a flight of stairs and fractured his nasal bone with concussion to his brain. After a short period he developed marked signs of acute mastoiditis, and, after due diagnosis was made, a left simple mastoidectomy was performed. Two months after his admission he was discharged recovered.

Miller and Lauppe[24a] reported only 21 cases in all literature since 1882, of the occurrence of mastoiditis following fracture of the skull. Although Guadjian[24b] reported a series of 2600 patients who suffered from fracture of the skull (Receiving Hospital, Detroit), and of this number 476 had bleeding or cerebrospinal fluid leakage from either one or both ears; and where cerebrospinal fluid leaked in ears there was a 50 per cent mortality.

Friesner and Rosenwasser[24c] think it not advisable to operate on such patients who show no signs of an extension of the infection beyond the confines of the labyrinth itself in those cases who suffer from labyrinthitis after an attack of chronic suppuration of the middle ear. They also feel that the laby-

rinthitis which is related with chronic suppuration of the middle ear which occurs either in the course of the disease or following a radical operation on the mastoid and belongs to a group which is more innocuous than either traumatic labyrinthitis or those which complicate acute suppurative otitis or mastoiditis. They are also of the opinion that in this group careful observation is necessary to decide which type will heal spontaneously and which will require intervention in serious types of labyrinthitis. The attitude of most is that in labyrinthitis, as in other pathological states, clinical phenomena are more valuable than laboratory data as indications for operation.

In a more carefully detailed analysis of the 3225 cases of both acute and chronic mastoiditis which I studied, I observed some very interesting data. In[24d] the series of acute mastoid patients with complications, the writer noted that four occurred between three months and six months of age; four cases were found to be between six months and one year; seven between two and four years; 12 ranged from four to six years; nine between six and eight years; 16 between the ages of 15 and 25 years; 27 were in the group between 25 and 35 years; seven between 40 and 50 years; 12 were from 50 to 60 years; three were found to be from 60 to 65 years, and two were in the age of from 70 to 75 years. In this group 70 were born in the United States, eight were of Italian birth; seven Russians, five Irish, three Spanish, four were born in Puerto Rico, one was born in Scotland, one British, one Newfoundlander, four Germans, two Chinese, one Central American, one South American, three Austrians, three were born in Poland and five were unknown.

The onset in this series of acute mastoids with complications also showed a varied result. Six patients had a sudden onset of between one and three days; five started between three and five days; and seven had an onset from three to six days; 12 were between seven and eight days in starting; four started between eight and 10 days; 11 had an onset between 11 and 15 days; 19 started between 16 and 21 days; (in this group one had an injury to the head following an automobile accident, and one had a discharging ear for 30 years with an acute flare-up; another was admitted in coma with his left ear full of pus, and one 10 days previously went swimming and then developed a marked headache accompanied by ear infection).

Fifteen patients had an onset between 22 and 30 days; 10 started after five to 12 weeks' onset; three started between 12 to 20 weeks; and two had an onset after a period of from five to 10 years.

Analyzing the occurrence of the side of the lesion in the acute infections with complications, I found 39 right acute mastoids; 46 left acute mastoids; 16 bilateral mastoiditis; four left chronic mastoids; two right chronic types, two recurrent infections and six which had acute mastoiditis but which were not operated on at all for special and indicated reasons. It was further found that the mortality in acute mastoiditis with complications varied in both sexes. Forty-six fatal cases in the males and 29 female fatal cases; and 31 male patients recovered with 18 of the females recovering.

There were a number of operations which were performed on the acute mastoid cases with complications thus:

Right simple mastoidectomies	45
Left simple mastoidectomies	44
Opening of sinus	15
Right jugular ligation	13
Left jugular ligation	25
Exploratory brain abscess (cerebellar)	7
Frontal brain abscess, exploratory	1
Right radical mastoidectomies	4
Left radical mastoidectomies	3
Secondary mastoid operations	5
Resection of old mastoid wound	2
Bilateral mastoidectomies	13
Right labyrinth operation	1
No operation	8
Cervical abscess	1
Right modified mastoidectomy	1

The author further studied the series of the chronic mastoid infections with complications and the following were concluded: The age occurrence showed that the youngest was six months old, and the oldest was 73 years of age; 1 was one year old; two were from two to four years; three were from four to six years; five numbered from six to eight years; five were in the group of from eight to 10 years; nine were from 10 to 15 years; 18 were between 15 and 25 years; 17 were between 25 and 35 years; 11 varied from 40 to 50 years; three were from 50 to 60 years; two between 60 and 65 years, and one at the age of 73. In this same group were 49 Americans; four of Italian birth; five Russians, and three born in Ireland; one

Spaniard; one whose nativity was in Greece, and one Hollander; one Armenian and one born in Finland; two could not be obtained. The onset in this series varied considerably. Since some of these cases came to the hospital in an urgent state and in certain instances some were brought in complete coma and stupor, a comprehensive history was not obtainable. A history was obtained in one case where the onset was sudden, and in two hours; in nine patients the onset was from one to seven days; in four other instances, (one case had an onset of three days prior to admission to the hospital, one seven days before and two, two weeks prior to admittance). In five cases the onset was from 15 to 30 days before being admitted for observation. The others varied in onset from three months to 25 to 30 years thus: Two patients had an onset from two to three months; two from three to four months; one from two to six months; two from 11 months to one year; two from one year to two years; 10 from two years to five years; four from five years to 10; six from 15 to 20 years' onset; two from 20 to 25 years; and one from 25 to 30 years' onset. Regarding the sex and the mortality, I found that there were 36 mortalities among the male patients, and 14 deaths in the female group with 21 males recovering and six female recoveries.

The above series of the chronic mastoids with complications had disclosed the following concerning the number and types of operations performed:

Right simple mastoidectomies	8
Left simple mastoidectomies	8
Opening of sinus	2
Right jugular ligations	7
Left jugular ligations	3
Exploratory—epidural abscess	1
Exploratory left cerebellar brain abscess	6
Exploratory temperosphenoidal brain abscess	5
Revision of mastoid	1
Right radical mastoidectomies	11
Left radical mastoidectomies	13
Right secondary mastoidectomies	1
Resection of old mastoid wound	1
Bilateral mastoidectomies	2
(One complicating tubercular meningitis.)	
Right labyrinth (Neumann's operation—drainage of the labyrinth)	1
Left labyrinth operations	2
Obliteration of the sigmoid sinus	1
No operations performed	5
(One patient operated on had diabetes.)	
(One case operated on for appendicitis, but who came under observation with an ear condition, with meningeal complications.)	

Little did Petit,[25] in the early part of the 18th Century, know that his first operation performed on an infected mastoid would set a new pace for the future of the specialty of otology. His operation was incomplete, yet it was done by making a small drill hole through the cortex. In spite of the unsuccessful attempt on his part, Schwartze,[26] in 1873, was not discouraged by the unpopular reception of this operation. He proceeded to perform a systematic method of removing the mastoid cortex and breaking down the infected cells. This procedure really created a new epoch in mastoid surgery.

In an analysis of 25 cases, an attempt was made to study the changes that take place within the mastoid process on successive days following the onset of acute otitis media. In[27] this series, A. P. Tibbets, Washington, D. C., emphasizes many practical points which are of importance, *viz.:* In acute mastoiditis, marked pathological changes may be present in the mastoid as early as the third day following the onset of an acute suppurative otitis media. The rapidity of the bone destruction depends largely upon the patient's age, as it is more rapid in younger patients, as well as upon the severity of clinical symptoms. Four out of five complications occurred in patients with sclerotic mastoids. This type of mastoid can be diagnosed by means of an early X-ray study and seems to point towards an early operation. Some cases of acute labyrinthitis, Gradenigo's syndrome, and meningeal irritation may recover without other interference than a simple operation. A sudden decrease or cessation of discharge from the middle ear, in presence of free opening of the drum and with an exacerbation of clinical symptoms, nearly always means complete blocking of the mastoid antrum which can be only relieved by operation. Patients who continue to show signs of mastoid involvement as late as the 18th day following the onset of an acute suppurative otitis media, even without marked evidence of bone destruction as shown by X-ray, have sufficient pathology within the mastoid to make satisfactory recovery without operation extremely doubtful.

In Tibbet's series of cases, 16 were acute which all began with well defined symptoms of mastoid involvement and had shown but little improvement, or had developed more unfavorable symptoms at the time of operation. Four of this group of 16, showed softening of the periosteum and cortex, and four other of the series had sclerotic mastoids. Of these four

cases there were three complications which were perisinus abscesses, and lateral sinus thrombosis, and two localized meningitis. In the nine remaining cases, the subacute group comprised of those which were operated upon from the 18th to the 55th day. These had shown a steady improvement and were considered to have a good chance for recovery until sudden unfavorable symptoms appeared which made operation imperative.

Kriegsman[28] analyzed 241 antrotomy cases and 86 per cent of radical mastoid patients. His conclusion is that in uncovering the sigmoid sinus, it is apt to invite a hazard more frequently than is generally accepted. He also noted that early operated cases are the most liable to undergo this complication; and he urges us to refrain from the liberation of the sinus in uncomplicated mastoids. In chronic cases, those in the stage of acute exacerbation appear the most dangerous. He thinks that exposure of the vein, without well founded suspicion for disease should be discarded.

Johnson,[29] in his study of 100 consecutive mastoidectomies, found that out of that number two deaths occurred — two septicemia on the fifth postoperative day. In his survey 60 per cent of patients were without complications; 4 per cent were complications of otitic origin before mastoidectomy and 36 per cent were complications that developed after operation. Four of this group were of a serious nature; an elevation of the temperature curve was the first warning of an existing complication in more than 86 per cent of these cases.

Effler reports a case of a female patient, age 40 years, who died from a chronic suppurative mastoiditis, with a past history of left mastoid operation at the age of 1 year.[30] The wound healed, but the left ear continued to discharge for about 39 years. The patient died at the operation with a diagnosis of acute chronic suppurative mastoiditis (left ear), with suspicion of left lateral sinus thrombosis. At autopsy, it was discovered that the lower lateral aspect of the mastoid tip was completely destroyed; the mastoid labyrinth was found completely exenterated and filled with fresh blood and foul smelling pus. A large perforation was found in the lateral sinus and from this opening all preoperative and operative profuse bleeding took place. In the jugular vein was found a thrombus about two inches above the point of ligation.

Clarence H. Smith[31] presents in literature a patient, age 12 years, who developed a right simple mastoiditis (acute), after scarlet fever six years prior to the recurrent mastoiditis with petrositis, temperosphenoidal abscess and with large epidural abscess. He further reports that the symptoms were very severe in this type of petrous pyramid inflammation. The epidural abscess was re-explored, localized, and drained with complete recovery.

In scarlet fever, complications occur in about 40 per cent of cases, and otitis media is a common complication. Involvement of the mastoid process on one or both sides is not an infrequent sequel to infection of the middle ear in scarlet fever.

MORTALITY OF MASTOIDITIS.

Bergh reviews the cases of otitis media occurring in 2425 patients suffering from diphtheria observed from 1921 to 1930. Acute disease of middle ear occurred in 121 patients. Of this number 61.2 per cent were one-sided. About[31a] one half of the conditions of otitis occurred after the 21st day of diphtheria. The largest number of patients having diphtheria were between the ages of from 6 to 8 years. It was found that nasal diphtheria is considered more ominous from the standpoint of otic complications than is the similar condition of throat diphtheria. It was also disclosed that the appearance of this primary disease of diphtheria had no influence on the mortality, but that there was a certain parallelism between the mortality rate and the frequency of complications in the middle ear. About two-fifths of the patients with diphtheritic otitis have an afebrile course. The prognosis of this type of otitis is good in regard to life and to mastoid complications. By all means, attempts should be inaugurated to prevent complications in the middle ear during the invasion of diphtheria by extreme precautions towards the cleanliness of upper respiratory tract and particularly should this be demonstrated towards the nasal chambers.

Twenty-five years ago Dr. Edwin A. Griffin[31b] noted that in a majority of hospitals of his city the tendency was to perform mastoidectomies at night. These were then known as "stat operations," as he terms it. In recent years the mortality has decreased, as more study has been given acute mastoid cases as compared to the last decade. Where an acute mastoid must

be operated, it is best performed not later than between the fourteenth and twenty-first day following the onset, as there will be less chance for complications to set in during this stage. He thinks that X-ray interpretations are only significant when rendered and plates taken by expert Roentgenologists; and, that where necrosis or destruction of the trabeculae in the acute mastoid process exists, it would be a good indication for surgical interference coupled with clinical indications. He routinely removes adenoids (except in presence of acute nasal infection) in all of his simple mastoid patients in childhood and does not hesitate to reincise the drum, except when an active postnasal infection exists. In acute middle ear infections he prefers early drum incisions as a preventive measure with a routine smear for culture.

The Gradenigo syndrome should be an indication of venous engorgement of the group of tympanic veins emptying into the inferior petrosal sinus. The syndrome of temporomaxillary orbital pain, trismus and edema of the lower lid, is of similar relationship with the Gradenigo syndrome but represents venous engorgement of the veins of the tympanic cavity draining anteriorly into pterygoid plexus and middle meningeal vein. Free bleeding from the middle ear by an early incision will create relief from existing symptoms and prevent suppuration of the petrous pyramid, but will not relieve advanced involvement of the petrosa with suppuration and bone coalescense.

Salissbury,[32] MacNalty and Malloch, quoted Rockemann and Brock who reported 705 cases of influenza and noted that 28 otitis media complications of this series had a hemorrhagic inflammation and a large amount of fibrin present. Mastoiditis is a frequent further complication, and this may occur before symptoms of otitis media have been observed. They noted that at a Base Hospital with British Expeditionary Forces in France, the number of patients with brain abscess occurring after the beginning of the pandemic of influenza was much greater than three years previous to the outbreak. Permanent unilateral deafness due to the involvement of the cochlear nerve has also been described.

Foci of infection of the mastoid may be present and an offensive odor of the discharge sometimes indicates caries of the bone. Streptococci, pneumococci, and staphylococci are pre-

dominant organisms found in this foci, but streptococci organisms are the most frequent of all.

I agree with F. L. Hoffman as regards the significant value of the statistical method in medicine: It has been of such exceptional value when applied with skill to the study of questions usually involved, because of innumerable factors underlying observed phenomena more or less indefinitely termed disease and death. The[33] fundamental law of statistics is the law of large numbers, and the accuracy of statistical data is in proportion to mass of material considered and the thoroughness of the methods of analysis in matters of detail. The exceptional cases are obliterated in the large number, which approaches more closely to the truth, and such require acceptance as approximately conclusive.

The U. S. Health Department, and vital statistics of various cities in this country and Europe, also Asiatic cities, since 1921, coded according to the manual of the International List of Causes of Death, and Title 89 of this manual reads, "Diseases of the Ear and of the Mastoid Process." Under this caption, 89-a—reads: Diseases of the Ear"; and under 89-b, "Diseases of the Mastoid Process." It appears that prior to 1921, all diseases of the ear and mastoid process were listed under 89 of the International List, and no attempt was made at differentiation between diseases of the ear and the diseases of the mastoid process. This will be seen in the listings which I present in this manuscript of various cities for the past 20 to 30-year periods. It also appears that in the earlier years, that is prior to 1915 to 1921, specific diagnosis as regards the actual cause of death was not listed in many cities because of the mixed classification in the various institutions and cities over the world.

I am attempting to present in this paper a general comparison of mortality statistics not only as related to some individual institutions, but also to compare various cities. By doing this I will show in concrete figures that the diseases of the ear and the mastoid process are not only on the increase, but that the mortality rates are also on the upward trend. The complications of the cerebral parts are more numerous in the last few years than was experienced heretofore.

The chart of the registration area in continental United States shows the causes of death from the mastoid process

is on the increase, which, in 1921 totaled 1060, and in 1931, the deaths aggregated 1718—a difference of 658 more than in 1921 (see Fig. 2).[24] In 1930 the total death rates from the diseases of the ear and the mastoid process amounted to 3946, out of which number 2348 were listed under the diseases of the ear and 1598 were under the mortality list of the diseases of the mastoid process. In 1931 the mortality figures for the combined disease listing was even greater, 4112, and in the caption of diseases of the ear, 2399, and diseases of the mas-

DEATHS — BALTIMORE.

Year	Diseases of the ear	Diseases of mastoid process
1901	9	—
1902	7	—
1903	13	—
1904	8	—
1905	13	—
1906	11	—
1907	12	—
1908	10	—
1909	9	—
1910	16	—
1911	17	—
1912	9	—
1913	19	—
1914	16	—
1915	15	—
1916	16	—
1917	17	—
1918	8	—
1919	22	—
1920	16	—
1921	10	—
1922	9	1
1923	23	—
1924	15	1
1925	20	5
1926	20	11
1927	23	17
1928	13	10
1929	22	4
1930	14	8
1931	20	12
1932	21	11

Fig. 1.

toid process 1713. In 1932, however, the aggregate figures for the combined diseases was listed 3841, and 2307 deaths for the diseases of the ear, and 1534 deaths for the diseases of the mastoid process. The average mortality rate for the three years, 1930, 1931, and 1932 was given as 3.33 rate per 100,000 estimated population of the United States. For the disease of

the ear alone, 2 per 100,000 estimated population, and for the diseases of the mastoid process for the corresponding years was 1.33 per 100,000 estimated population of this country. The author feels that such high mortality figures for this disease is entirely too much, especially, when the specialty of otology is rapidly gaining more scientific background. Too many so-called and self-styled otolaryngologists, who have had no training at all in the field of otology, but who have entered and attempted to practice the specialty in recent years, has played some part in the increase in the mortality figures of ear diseases and the mastoid process.

New York State,[35] exclusive of the city of New York, gives the annual report in 1932 under the diseases of the ear and

THE REGISTRATION AREA IN CONTINENTAL UNITED STATES.

Year	Per cent of total population	Deaths from mastoid process	
		No.	Rate per 100.000 pop.
1931	96.3	1718	1.4
1930	96.2	1605	1.4
1929	95.7	2035	1.7
1928	95.3	1783	1.6
1927	91.5	1505	1.4
1926	90.1	1581	1.5
1925	89.6	1328	1.3
1924	88.4	1223	1.2
1923	87.7	1295	1.3
1922	85.4	1081	1.2
1921	82.3	1060	1.2

This cause was not shown separately prior to 1921 U. S. census.

Fig. 2.

the mastoid process, the mortality figures for that year being 186; the diseases of the ear alone 119, and the diseases of the mastoid process death rate being 67 for that year. The analysis further explained the age groups for that year; the mortality for the combined ear diseases and the mastoid process was registered as 35 cases from 1 to 4 years of age patients; from 5 to 9 years of age, the deaths given were 26; 10 to 14 years, the total number was 20; from 15 to 19 years, 14; 20 to 24 years, 2; 25 to 29 years, 12; 30 to 39 years, 19; 40 to 49 years, 20; 50 to 65 years, 25; 65 to 74 years, 9; 75 to 79 years, 2; and 80 to 84 years, 2. Out of the total number of 186 deaths 199 were males and 67 were female patients.

From 1901 to 1921, the city of Baltimore classified the mortality figures of ear conditions under "The Diseases of the Ear" and the total for the 20 years were 288. Beginning in

1922, up to 1932, a period of 11 years, the total death rate from the combined diseases of the ear and the mastoid process aggregated to 200 or at the rate of 10 deaths each year for the average population of about 700,000 people. Eighty deaths were given for the same 11-year period for the diseases of the mastoid process (see Fig. 1).[36]

The president of the board of health of the city of Chicago sent me no information related to statistics from 1901 to 1911, but beginning with 1912, up to 1920, the total mortality

CHICAGO BOARD OF HEALTH, BUREAU OF VITAL STATISTICS.
Mortality Statistics of Diseases of Ear and Mastoid Process.

Year	Ear	Mastoid	
1901			No information
1902			
1903			
1904			
1905			
1906			
1907			
1908			
1909			
1910			
1911			
1912	70	—	
1913	77	—	
1914	40	—	
1915	48	—	
1916	50	—	
1917	54	—	
1918	52	—	
1919	75	—	
1920	89	—	
1921	113	33	
1922	65	31	
1923	101	64	
1924	89	36	
1925	115	28	
1926	94	41	
1927	83	46	
1928	97	46	
1929	99	44	
1930	85	44	
1931	92	34	
1932	103	39	

Fig. 3.

for diseases of the ear and mastoid process was 688. From 1922 to 1932, a 10-year period, the total mortality from the diseases of the ear and the mastoid process was 1509 and those figures of the mastoid process alone for the 10-year period were 486. The population of the city of Chicago on the average for the past 10 years has been 3,332,276 (see Fig. 3).[37]

From 1901 to 1925, the total number of deaths in Philadelphia from the diseases of the ear and the mastoid process were 1487. Beginning in 1926 and ending in 1932 the mortality for diseases of the ear was reported 259, while the mortality figures for mastoid process alone were 415 for the same period from 1926 to 1932, inclusive. The population of Philadelphia, from 1920 to 1930, averaged 1,887,370, and the

DEATHS IN PHILADELPHIA FROM MASTOID DISEASES.
Years 1901 to 1932, inclusive.

Year	Diseases of the ear	Diseases of the mastoid process
1901	1	
1902	2	
1903	3	
1904	27	
1905	33	
1906	31	
1907	47	
1908	63	
1909	76	
1910	52	
1911	57	
1912	53	
1913	60	
1914	62	
1915	81	
1916	63	
1917	86	
1918	61	
1919	68	
1920	75	
1921	96	
1922	89	
1923	83	
1924	112	
1925	97	
1926	36	51
1927	43	40
1928	74	92
1929	32	60
1930	21	64
1931	29	63
1932	24	45

Fig. 4.

mortality for diseases of the ear averaged 2.1 per 100,000 population (see Fig. 4).[38]

New York City, with a population of 5,620,048 in 1920, presents an entirely different picture. From 1901 up to 1916, the total mortality for diseases of the ear was 2921, for a period of 16 years, or 182 every year. From 1917 to 1930 the figures were 4853 or almost twice that from 1901 to 1916, although the population was only 1,320,048 more on average

than in 1901-1916. From 1931 to 1932, inclusive, the report for diseases of the ear mortality was 320 and the diseases of the mastoid process mortality for the two-year period was given as 555. The population of New York City in 1930 was 6,930,446 (see Fig. 5).[39]

CITY OF NEW YORK.

Diseases of the Ear			Disease of the Ear	Disease of the Mastoid process
1901	81	1917	254	
1902	122	1918	261	
1903	147	1919	289	
1904	175	1920	287	
1905	153	1921	340	
1906	188	1922	417	
1907	201	1923	377	
1908	159	1924	389	
1909	142	1925	378	
1910	152	1926	357	
1911	246	1927	330	
1912	187	1928	412	
1913	269	1929	382	
1914	266	1930	381	
1915	249	1931	184	296
1916	184	1932	139	259

Fig. 5.

TOKIO, JAPAN.

Deaths Due to Mastoid Diseases per 100,000 of Population.	
1912	0.59
1913	0.60
1914	0.56
1915	0.70
1916	0.64
1917	0.67
1918	0.79
1919	0.76
1920	0.75
1921	0.83
1922	0.78
1923	0.88
1924	0.80
1925	0.78
1926	0.68
1927	0.69
1928	0.72
1929	0.72
1930	0.68
1931	0.78

Fig. 6.

In Tokio, Japan, the deaths due to mastoid diseases from 1912 to 1921, averaged .69 per 100,000 population; and from 1921 to 1931, the mortality figures given were .761 per 100,000

population, but no figures were given prior to 1912 as there was no such classification made. The population of Tokio in 1930 was 2,070,529 (see Fig. 6).[40]

In contrast to New York City, the city of Los Angeles presents an interesting comparison. In 1906, in the city of Los Angeles, when the population was 240,000, no deaths were recorded for ear conditions, also no deaths in 1909 and 1910. From 1906 to 1925 the total mortality was recorded as 99. In 1925, although the population was 1,014,622, there were only 12 deaths due to ear diseases. In 1926 the city of Los Angeles first began to classify the mortalities in ear diseases and subdivide the diseases of the ear from mastoid involvement. From the study of the above figures one would judge the low mortality due to the favorable climate of this city. Beginning with 1926, although the population varied little between

CITY OF LOS ANGELES.
From Diseases of the Ears.

		Population			Population
1906	0	240,000	1916	1	500,000
1907	1	263,782	1917	2	533,535
1908	2	295,687	1918	7	500,000
1909	0	307,322	1919	2	563,000
1910	0	319,198	1920	6	576,673
1911	1	347,550	1921	13	660,251
1912	1	427,000	1922	19	736,963
1913	2	430,000	1923	11	802,358
1914	1	438,914	1924	19	850,143
1915	2	475,367	1925	12	1,014,622

Deaths from Otitis Media (89a) and Disease of Mastoid Process (89b).

	89a	89b	Population
1926	15	11	1,056,983
1927	29	14	1,079,789
1928	40	23	1,152,806
1929	43	21	1,206,764
1930	17	6	1,238,048
1931	57	30	1,255,829
1932	42	20	1,283,859
1933	44	25	1,281,266

Fig. 7.

1,014,622 and in 1933, 1,281,266, still the mortality had been constantly on the increase. One would conclude that either the virulence of the organisms changed, or that methods in diagnosis have certainly improved in the past few years (see Fig. 7).[41] It may be, too, that Jean[42] was correct when he stated

that the climate of Iowa might be responsible for increased ear infections.

Toronto, Ontario, Canada, gives from 1909 to 1920, inclusive, death rate figures for the diseases of the ear and mastoid process, 446. Whereas, from 1921 to 1932 the mortality figures for diseases of the ear and mastoid combined was given at 1321. The population averaged from 1902 to 1920, about 415,000 annually, making it on the average of 9.25 death rate per 100,000 population for the 12-year period, 1909 to 1920; and 24.1 per 115,000 population on the average

ONTARIO.

Diseases of the Ear.

1909	41
1910	40
1911	48
1912	47
1913	37
1914	34
1915	24
1916	26
1917	30
1918	22
1919	28
1920	69

Diseases of the Ear and Mastoid.

	M	F	Total
1921	34	37	71
1922	37	31	68
1923	—	—	83
1924	—	—	80
1925	67	41	108
1926	64	50	114
1927	66	47	113
1928	63	50	113
1929	77	43	120
1930	68	47	115
1931	70	50	120
1932	77	49	126

Fig. 8.

annually, from 1921 to 1932, which is almost three times greater, although the population total increase averaged 112,000; this means that the high mortality rate for the diseases of the ear and mastoid process is entirely out of proportion to the comparative small increase of the population (see Fig. 8).[43] Here again the change of climate has much to do with the problem of mastoiditis and its mortality rates.

O. J. Dixon[44] quotes 30 cases of mortality in mastoiditis infection, 13 of which died of meningitis, eight of pneumonia, five abscess of brain, and four sinus thrombosis. He also studied 186 mastoidectomies performed at the St. Luke's Hospital, 1923-1928, and of this total number there were 13 deaths as follows: Two brain abscess, one sigmoid sinus thrombosis, six streptococcus meningitis, one septicemia, one diarrhea, one sinus thrombosis, one postoperative shock. Dixon also found that at the Isolation Division of the General Hospital, 1923-1927, inclusive, out of a total number of 1939 infectious diseases, two deaths resulted from a series of 12 mastoidectomies performed. He also found that at the 350-bed Kansas City General Hospital, where 60 mastoidectomies were performed between 1923 and 1928, six patients died; and out of a total of 17,891 patients admitted at St. Margaret's Hospital between 1923 and 1927, there were 140 ear cases, out of which number there were 77 mastoids, of which five were fatal; of this number three were caused by otitic meningitis, one by sigmoid sinus complication, and the other by septicemia.

Carlos Monserrat[45] made a bacteriological study of meningitis of 10,0000 autopsies, which were performed in the Philippines during a 16-year period, beginning in 1907 and ending in 1923. In this entire group he found only two cases which showed definite evidences of suppurative ears. Although he found out of the whole series 190 cases of suppurative types of meningitis, of which the most common organisms identified were pneumococcus, staphylococcus, streptococcus, B. pestis, B. influenza, pneumobacillus, and undetermined bacteria.

Renaud[46] found pus in the mastoid antra of 70 infants dying of gastrointestinal and nutritional disturbance. For years it was thought to be nutritional disturbance, but Marriott[47] thought actual treatment of the nose, throat and ear brought better results than changes in character of food. It was discovered that gastrointestinal symptoms frequently disappear following myringotomy. However, in some cases, Marriott found neither diarrhea nor otorrhea stopped and infants died. (This was his opinion in 1923.) In favorable cases falling of temperature was present and diarrhea stopped. Streptococcus hemolyticus organisms most commonly found etiological factor. Rosamond[48] feels that the pediatrist can be consulted with advantage concerning the type of anesthesia to be used; although, in measures that pertain to surgical relief of the

infection, and the conservation of hearing, the otologist is really paramount in his opinion. In the experience of Marriott,[47] he finds that over 85 per cent of all gastrointestinal and nutritional disturbances in recent years have been due to infections of the ear. nose and throat. It has been found, too, in those children who are debilitated over a period that the best results obtained as an anesthetic, in infants particularly, is nitrous oxide.

When a competent aural surgeon performs his work, the mortality figures are about[49] 1 to 2 per cent on a large series of patients. Lyman and Alden[50] had eight recoveries and seven deaths in the first series of their cases, whereas, in their last series, there was a lower mortality rate.

Early in the mastoid disease during infancy, edema may be due to seepage through the normal sutures. Traumatism of the canal which in infancy is membranous, and may even be pulled loose from its attachment to the drum membrane, may produce edema. In cases of uncertain diagnosis, time is an important element to decide between simple edema and tumor due to actual rupture of cortex. It must not be overlooked that when fever is properly studied and graphed, it lends valuable information giving intensity and degree of the invading disease. The drum, whether it is bulging, helps to give important information. The lymphatics are nine times more permeable in infants than in adults. Although persistent drainage can be obtained by repeated incisions of the drum if required, still, when there is constant downward bulging of the drum and canal wall, this can be the guiding mark for the most certain known indication of mastoid periostitis. When there is a profuse and continuous discharge, it is apt to show an otitis media with infection to embryonal tissue filling the cavity. A falling hemoglobin or red count shows that there is a downward constitutional trend. One out of three brought to operation show a large mastoid area when X-ray is taken before.

There may be types of mastoiditis which are essentially hidden. This condition is rare, but when it does present itself, usually in the form of an epidemic in certain regions and with the signs of only a sagging of the posterior superior part of the drum membrane; there is no other sign except cholera infantum syndrome, and its related symptoms.

Dr. Roth[50a] with whom I had been associated a number of years, stressed the importance of bacteriological examination in aural patients where thin, scanty discharge was present with an increasing deafness and absence of pain; such types were the clinical cases in which this bacterium was usually an indication for immediate mastoidectomy, regardless of X-ray disclosures. In adults, a persistent high septic temperature for a week following a primary acute ear abscess, he believed, is highly suggestive of thrombotic mastoiditis, and in the absence of other definite clinical entities, is an indication for opening the mastoid, even with a negative X-ray report. He also believed that anticipatory surgery was indicated where an increasing localized headache followed several days after a primary acute ear abscess, regardless of what the X-ray disclosed and provided there had been sufficient opening in the drum for adequate drainage of the middle ear region. In the event of a chronic discharging ear continuing after the mastoid was operated on in childhood, it is unwarranted to do a radical operation except for threatened intracranial complications; the risk of operative meningitis is greater here than in any other type of uncomplicated ear discharge. He further believed that osseculectomy should be more often tried for the arrest of chronic discharging ears, deferring and thus making radical surgery unnecessary in many instances. Often he found that the failure to distinguish neuralgic headache associated with chronic mastoiditis was not rarely the cause for inexpedient radical interference.

Moreau[51] reported five cases of mastoiditis in infants from 4 months to 2 years of age. Four had retroauricular abscess and one had otorrhea alone. In each case there was a postoperative rise in the temperature, dyspnea, and death. No explanation was given for the fatal outcome, but similar cases have been reported by others.

Retrauney[52] reported 30 cases of mastoiditis in infants operated on in Bordeaux Hospital. There occurred five deaths in this series.

Faunce and Shambaugh[35] are of the opinion that the best treatment of brain abscesses rests entirely on an early and proper diagnosis, including the recognized surgical intervention; whereas surgery, Dandy[54] thinks must not be considered in patients suffering from brain abscesses of acute nature.

Dandy is of the opinion that in any form of successful treatment related to brain abscess, it is essential to wait for encapsulation before operating.

Where labyrinth suppuration is suspected, surgical drainage is often a life saving procedure. Yet, every labyrinth suppuration is considered as possibly reparable as long as there is any cochlear or vestibular function remaining. The caloric test is the only means by which one can determine the true condition of the vestibular labyrinth. If[55] upon douching the ear with cold water or hot, nystagmus or vertigo can be elicited from any of the canals, operation on such a labyrinth is strongly contraindicated, even in the presence of total deafness.

The author feels that in trying to decrease the mortality and the morbidity, it is important to bear in mind the difference between a surgeon and an operator. We all know that the surgeon is always ready to deliberate if it is necessary to perform an operation, and if he does, it is usually with a careful and skillful understanding regarding the type of case in hand; and he also attempts to make the correct diagnosis before his knife begins to cut. Holt was once heard to express his opinion about 25 years ago, and he said if mastoiditis follows otitis complicating acute infectious diseases, early in childhood, they may recover without operation. Who knows how many operations are done too early, and how many patients suffering with acute mastoiditis are not recognized early enough; how many are operated on much too late when serious complications have set in beyond any medical or surgical help? The writer feels that, particularly these days of increasing threats and attempts on the part of numerous cults and highly commercialized mental healing organizations, the time is ripe to ward off criticisms from various channels, by attempting, at all costs, to help decrease the rising tide in mortality flows from various causes in the diseases of the ear and the mastoid process.

CONCLUSIONS.

1. The mortality rates of acute and chronic diseases of the mastoid are increasing entirely out of proportion to the increase in the population, in various cities in the United States.

2. Out of a total of 2100 acute mastoiditis cases admitted to Bellevue Hospital in a period of 21 years, about 16.66 per

cent developed cerebral complications; and out of 126 complications (16.66 per cent), there were 28.55 per cent recoveries and 71.45 per cent mortalities.

3. Out of 1125 chronic mastoiditis cases admitted to Bellevue Hospital in 21 years, there developed 83 cerebral complications, and out of this number, 66 cases were fatal, and 17 patients recovered.

4. Meningitis complicating acute and chronic purulent otitis media are invariably fatal; out of 55 cases of meningitis complicating acute otitis, 49 were terminated fatally; and out of 32 patients who suffered from meningitis complicating chronic otitis, 28 terminated fatally.

5. Brain abscess conditions in patients complicating acute otitis, are of very high mortality rates; out of 18 cases of brain abscess following acute otitic origin, 16 were fatal, two were nonfatal. Out of 15 brain abscess patients of chronic otitic origin, 11 were fatal, and four recovered.

BIBLIOGRAPHY.

1. KOVACS, ARTHUR: Otitic Generalized Infection, Origin, Management, Outcome. THE LARYNGOSCOPE, 373-379, May, 1934.

2. RIACH, FRANCES R.: Record Librarian from the Johns Hopkins Hospital, Baltimore, Md. (From official correspondence of Dr. M. Martyn Kafka.)

3. COATES, G. M.: Mastoid Infection in the Infant. *Amer. Otol., Rhinol. and Laryngol.,* 36:921, Dec., 1927.

5. PENFIELD, WILDER: Principles of Pathology of Neurosurgery, *N. L. L. Sys. of Surgery,* Vol. II, pp. 303-310.

6. WEED: 1917.

7. PENFIELD, WILDER: Principles of Pathology of Neurosurgery. *N. L. L. Sys. of Surgery,* Vol. II, pp. 303-310.

8. ALEXANDER, G.: Studies Concerning the Surgical Treatment of Otitic Meningitis, from Ear Dept. of General Polyclinic, Vienna. With translation by Geo. E. Davis. *Archiv. für Ohrenheil.,* Vol. 75, pp. 22; *Ann. of Otol., Rhinol. and Laryngol.,* Vol. 76, pp. 585-644, 1909.

9. DRUSS, JOSEPH G.: Labyrinthitis and Sinus Thrombosis Complicating Suppuration of Middle Ear. *Archiv. of Otolaryngol.,* Vol. XIX, pp. 671-683, June, 1934.

10. KAFKA, MARTYN M.: (Service of Bellevue Hospital, 1912 to 1933 inclusive; Otolaryngological Division, New York City.) (All divisions included).

11. OCHSNER'S Surgical Diagnosis and Treatment. Lea and Febiger, Phila.: 1:327, 1920.

12. KANAVEL, A. B.: Diagnosis and Treatment of Tumors, Inflammation and Abscess of Brain.

13. MACEWEN, WM.: Pyogenic Infective Diseases of the Brain and Spinal Cord. Macmillan, N. Y., 1893.

14. COLEMAN, C. C.: Reduction of Mortality of Brain Abscess by Simple Methods of Treatment. *South. Med. Jour.*, pp. 484-487, June, 1930.

15. Mortality of Brain Abscess. *Nelson's L. L. S. of Med.*, Vol. VI, p. 310.

16. HORROX, GILBERT: Surgery of the Brain. *N. L. L. Sys. of Surg.*, Vol. II, p. 367.

17. EAGLETON, W. P.: Operative Treatment of Cerebellar Abscess of Otitic Origin. *Surg., Gyn. and Obst.*, 39:653, 1924.

18. HORROX, GILBERT: Surgery of the Brain and Brain Abscess. *N. L. L. Sys. of Surg.*, Vol. II, pp. 371-374.

19. BRAUN, ALFRED: Purulent Diseases of the Meninges and the Brain. *N. L. L. S. Med.*, Vol. VI, Chap. XV, pp. 295-324G.

20. PENFIELD, WILDER: Principles and Pathology of Neurosurgery. *N. L. L. Sys. of Surg.*, Vol. II, pp. 303-310.

21. KOVACS, ARTHUR: Otitic Generalized Infection, Origin, Management, Outcome. THE LARYNGOSCOPE, pp. 373-379, May, 1934.

22. HAYS, HAROLD: Unusual Complications, Lateral Sinus Thrombosis. *Ann. of Otol., Rhinol. and Laryngol.*, Vol. XLI, No. 2, pp. 584-593, June, 1932.

23. GRANT, FRANCIS C.: The Mortality from Abcess of the Brain. *Jour. Amer. Med. Assn.*, Vol. XCIX, No. 7, Aug. 13, 1932.

24. BRAUN, ALFRED: Pathway of Infection to Brain. *N. L. L. Sys. of Med.*, Vol. VI, pp. 295-310.

24a. MILLER and LAUPPE: *Jour. of Mich. State Med. Soc.*, 24:952-914, Dec., 1930.

24b. ERSNER, MATTHEW S., and MEYER, D. S.: Cerebrospinal Otorrhea. THE LARYNGOSCOPE, Vol. XIV, No. 8, pp. 668-674, Aug., 1934.

24c. FRIESNER, ISIDORE, and ROSENWASSER, HARRY: Treatment of Labyrinthitis. *Arch. Otolaryngol.*, Vol. 20, No. 2, pp. 140-151, Aug., 1934.

24d. KAFKA, MARTYN M.: Service of Bellevue Hospital, Oto-Laryngology.

25. SEELIG: History of Medicine. *Petit J. L.*, pp. 148-150.

26. SCHWARTZE: *Archiv. für Ohrenheil.*, 2, 1873.

27. TIBBETS, A. P.: Gross Pathological Changes Following Onset of Acute Suppurative Otitis Media. THE LARYNGOSCOPE, May, 1934.

28. KRIEGSMAN, G.: Zur Frage der Post-Operativen Sinus Thrombose. *Archiv. für Ohren, Nasen- und Kehlkopf.*

29. JOHNSON, HENRY P.: Study of 100 Consecutive Mastoidectomies, Maine General Hosp. Temperature After Mastoidectomy. *Arch. of Otolaryngol.*, Vol. 19, pp. 660-670, June, 1934.

30. EFFLER, LOUIS R.: Suppurative Perforation of Lateral Sinus With Preoperative and Operative Hemorrhage. THE LARYNGOSCOPE, pp. 228-231, March, 1934.

31. SMITH, CLARENCE H.: Recurrent Mastoiditis With Petrositis, Temperosphenoidal Abscess, Large Epidural Abscess and Recovery. THE LARYNGOSCOPE, pp. 221-222, June, 1934.

31a. BERGH, E.: Acute Purulent Otitis Media With Diphtheria. *Monatsschr. f. Ohrenheil.*, 68:1, Jan., 1934.

31b. GRIFFIN, EDWIN A.: Brooklyn.

32. SALISSBURY, A., MACNALTY and MALLACH: In 705 Cases of Influenza. Rockmann and Brock.

33. HOFFMAN, F. L.: Text Book Statistics.

34. KAFKA, MARTYN M.: Official Correspondence.

35. KAFKA, MARTYN M.: *Ibid.*

36. KAFKA, MARTYN M.: *Ibid.*

37. KAFKA, MARTYN M.: *Ibid.*

38. KAFKA, MARTYN M.: *Ibid.*

39. KAFKA, MARTYN M.: *Ibid.*

40. KAFKA, MARTYN M.: *Ibid.*

41. KAFKA, MARTYN M.: *Ibid.*

34. Statistics from U. S. Bureau of the Census, Dep't. of Commerce, Wash. D. C.; Murphy, T. F., M.D., Chief Statistician for Vital Statistics. Feb. 5. 1934, Statistics from 1921 to 1931.

35. New York State Annual Report, 1932. Dep't. of Health, State of N. Y. Vital Statistics.

36. Health Dep't., City of Baltimore, Reports 1901-1932. Mortality of Mastoiditis and Diseases of Ear. Huntington, Williams, M.D., Comm. of Health.

37. Health Dep't., City of Chicago, Reports 1901-1932. Diseases of Ear and Diseases of Mastoid Process. Bundensen, Herman N., President, Board of Health.

38. Dep't. of Health, City of Philadelphia, Reports 1901-1932. Diseases of Ear and Mastoid Process, Mortality. Cassedy, Harry W., Registrar.

39. Dep't. of Health, City of New York, Reports 1901-1932. Diseases of Ear and Diseases of Mastoid Process, Mortality. Wynne, Shirley, Comm. of Health.

40. Central Sanitary Bureau, Home Dept., Tokio, Japan. Reports 1912-1931. Diseases of Mastoid and Diseases of the Ear. Ohshimg, T., Director of Sanitary Bureau, Home Dep't.

41. Dep't. of Health, City of Los Angeles, Cal. Reports 1906-1933. Diseases of Ear and Mastoid Process, Mortality. Stevens, Geo. M., M.D., Ass't. Health Officer.

42. JEAN, P. C.: Upper Respiratory Infections. *Jour. Amer. Med. Assn.*, 87:220-223, July 24, 1926.

43. KAFKA, MARTYN M.: Personal Correspondence from Dep't. of Provincial Secretary of Toronto, Ontario. Reports 1909-1932. Diseases of the Ear and Mastoid Process, Mortality. Manchester, S., Director of Vital Satistics.

44. DIXON, JASON O.: The Causes of Death in Mastoiditis.

45. MONSERRAT: Bacteriological Study of Meningitis in Philippines, Based on 10,000 Autopsies.

46. RENAUD, MAURICE: *Bull. et Mem. Soc., Med. de hop. de Paris,* 45:326-1352-1354, 1921.

47. MARRIOTT: Observations Concerning Nature of Nutritional Disturbance. Amer. Ped. Soc., 1925.

48. ROSAMOND, EUGENE: When Shall We Operate on an Infant Mastoid?

49. KERRISON, P. D.: Diseases of Ear. J. B. Lippincott Co., Phila., p. 188.

50. LYMAN, H. W. and ALDEN, A. M.: Gastrointestinal Disturbances in Infants as a Result of Abscess Infection in Mastoid. *Trans. Amer. Laryngol., Rhinol. and Otol. Soc.*, 1925-1927.

50a. RUTH, AARON: Brooklyn.

51. MOREAU: De la Granite des Operations Mastoidunnem Chez le Nourrisoun. *Rev. de Laryngol.*, 47:667, 1926.

52. RETRAUNEY, H.: Reports 30 Cases of Mastoiditis in Infants, Operated on in Bordeaux Hosp. Mastoitite du Nourrisson et Mort en Hyperthermie. *Rev. de Laryngol.*, 50:164, 1929.

53. FAUNCE and SHAMBAUGH: *Arch. Laryngol.*, Vol. XVII, No. 5, p. 673, May, 1933.

54. DANDY, W. E.: System of Surgery. Dean Lewis, Vol. XII.

55. FISHER, LEWIS: Evaluation of Caloric and Rotation Tests. *Ann. Otol., Rhinol. and Laryngol.*, Vol. XV, No. 4, Dec., 1931.

807 St. Marks Avenue.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SECTION OF OTO-LARYNGOLOGY.

Meeting of Jan. 11, 1935.

(Continued from August issue.)

DR. J. S. REYNOLDS reported two cases of "Diverticulum of the Esophagus."

Mr. J., age 67 years, had no difficulty in swallowing, but complained of distress in the neck after eating, with some regurgitation of food. He had a diverticulum about two inches in length and about three-fourths of an inch in diameter as shown in the X-ray.

Mrs. B., age 66 years, stated that for the past four months she had had trouble in swallowing solid food. There was a large palpable gland on the left side of the neck. X-ray findings proved conclusively that there was no malignancy present, but that she had a diverticulum of the esophagus one inch in length and one-half inch in diameter.

Both of these diverticula had their origin in the upper end of the esophagus. As yet no operation has been done.

DR. ISODORE FRIESNER was the guest speaker of the evening, and gave a talk on "Pathways of Intracranial Extension of Suppurative Ear and Mastoid Disease." This was illustrated with numerous lantern slides, and slides of microscopic sections.

Dr. Friesner said he had come to present the results of their investigations, to state some of their problems, to state also what solutions had presented themselves to him, and to have the members discuss these with him. About eight years ago one of the young graduates of the ear service went to Vienna for special study for about a year. He said they were able to obtain about 90 per cent of autopsies at Mt. Sinai Hospital in New York, and much of their work was the result of routine pathological examinations. As a result they had amassed a large amount of pathological material from their cases. These were not cases that came to the hospital on the otologic service; they were for the most part patients operated on elsewhere. They had 24 pathological specimens of petrositis, and of these 24, 13 came to the hospital with intracranial complications; and 11 of these had developed meningitis under their care in the last seven or eight years. The extension to the intracranium, either the brain or meninges, is always due to an osteitis. In 19 of their 24 cases of osteitis the infection had spread along the superior and posterior perilabyrinthine cells; in 12, the supralabyrinthine route was the more marked.

DISCUSSION.

DR. CARL LARSEN said he must admit that he was just a little confused as to what Dr. Friesner considered the most common avenue of infection in these intracranial complications, but he gathered from the speaker that infection by way of the petrous pyramid was very common. It has been Dr. Larsen's experience that a great many cases of meningitis, and probably two-thirds of all cases of brain abscess, are infected by way of the lateral sinus.

Dr. Larsen cited a case which he had seen in consultation recently. The patient, a boy, age 8 years, had pneumonia for two weeks and acute otitis media of both ears for about the same length of time. According to his physician, the left ear was dry and the right ear was discharging only slightly. He did not have a high temperature at any time, nor was it of the sinus thrombosis type. The doctor had ascertained no brain symptoms of any kind. That morn-

ing the patient had had a light breakfast; was seized with vertigo and vomiting and, when his physician saw him an hour or two later, he was having convulsions. When Dr. Larsen saw him he was in extremis, unconscious, had marked twitching of both sides of the face, particularly of the right, and had a marked horizontal and rotary nystagmus to the affected side. He died in a few hours. Autopsy showed a ruptured cerebellar abscess on the right side. The mastoid showed pus and the lateral sinus showed a large septic thrombus and was filled with pus. Culture showed streptococci.

Dr. Kenneth Phelps said Dr. Friesner had made a statement concerning a type of mastoiditis called hemorrhagic. He wondered if Dr. Friesner would elaborate a little further as to why he does not believe there is such a condition as hemorrhagic mastoiditis.

Dr. Friesner said, of course there can be cases of sinus thrombosis without an osteitis in the pyramid; they can be directly through the wall of the sinus; there are cases of phlebitis in which the infection passes through the pyramid. He had seen one of these in which he could trace the infection about 1 cm. away from the posterior lacerated foramen and in the sinus there was a purulent thrombus not larger than a pea. Some one asked Dr. Friesner why he did not believe there is such a thing as hemorrhagic mastoiditis. He could divide his reasons into two parts. First, the pathological side. The statement had been made that in acute hemorrhagic mastoiditis the pathological picture is as follows: submucous infiltration, thrombosis in the veins and no bone disease. He said he was very much interested because he had never seen such a case. And he thought it very bad psychology, if it were not true, to put a pathological and clinical picture of this type in the minds of young men beginning otology for the reason that if one operated on a case in which there was no osteitis and no abscess formation and found an acutely congested mastoid, under such conditions one would ask one's self what was there about this case that made me operate so early, because obviously the case should not have been operated so early. He said he had talked to pathologists in Vienna — to Neumann, to Alexander, to Ruttin, to Fischer — and he had asked them if they recognize such a condition. Of course, these men are masters in histopathology, and they denied it, saying there was no such thing.

Dr. Friesner said he felt that the picture described as hemorrhagic mastoiditis is a very early infection and possibly is capable of resolution. It is the type of infection that general surgeons see with infection in the arm — redness and swelling, but no localization. And he also felt that the principles of general surgery apply to otologic surgery as well; he said he doubted very much whether a general surgeon would operate on such a pathological picture in the soft parts. Dr. Friesner said he had been fortunate enough to speak in a great many communities and he did not believe there is any group of surgeons among whom there would be any who would deny the existence of an acute appendicitis or an acute osteomyelitis of one of the long bones; but in every community where he had been he would find a few men who believe in acute hemorrhagic mastoiditis, and, on the other hand, many men who denied its existence. Were acute hemorrhagic mastoiditis a reality it would be recognized by all otologists today.

Dr. W. E. Camp said he would like to compliment Dr. Friesner on this presentation and ask him a few questions. In studying the slides several questions had occurred to him. First, whether the anatomical "make-up" of the mastoid had any bearing on the probability of intracranial complications; and, second, to ask him concerning the question of hemorrhagic mastoiditis. One does see cases of acute fulminating mastoiditis in which there is very little necrosis of the bone and very little pus in the mastoid cells. He recalled a case he had not long ago in which there was free pus in the cells and very little bone necrosis. Dr. Camp was of the opinion there must be some term applied to these acute fulminating cases in which this condition is present.

Dr. V. J. Schwartz said he would like to ask Dr. Friesner one question. Some time ago he had reported to the Academy a case involving a woman who

lived out of the city (but whom he had known previously), who developed an acute otitis. She had consulted Dr. Schwartz after she had apparently recovered from the acute effects of her illness, and gave a history indicating that she had had an acute diffuse suppurative labyrinthitis. Then arose the question as to what to do in the case. She was in the stage in which she had practically no symptoms except that she could not hear. Dr. Schwartz said he was rather inclined to advise operation, but it isn't so easy to transmit the idea of an operation of that kind to a patient who is almost certain she has recovered.

Dr. Friesner (in answer to Dr. Camp) felt that was not a case of acute hemorrhagic mastoid; it had pus in it. That is a suppurative mastoiditis, rather early; but it is not the classical hemorrhagic mastoiditis. Dr. Friesner said he had had no experience with acute hemorrhagic mastoiditis. He had seen or had had reported to him somewhere between 3000 and 3500 cases of acute mastoiditis. In all this material there had been no case of acute hemorrhagic mastoiditis. The supposition is that most of these cases are due to hemolytic streptococcus.

Dr. Friesner (in answer to Dr. Schwartz) said he would have left that woman alone. He still had the manuscript of a paper he had written on labyrinthitis; and the type of labyrinthitis Dr. Schwartz described looked to him like the type associated with latent mastoid with no symptoms. It is quite possible that woman may have had latent symptomless trouble in her mastoid that could have been suspected only by her poor hearing, something about the appearance of the ear that meant it was not resolving after three or four weeks, or some insidious infection that did not give frank signs of mastoiditis. If one does get a labyrinthitis in this type of case it is usually due to an erosion in the semicircular canal and that type of labyrinthitis usually has a fatal outcome, unless one attacks it immediately. But Dr. Friesner said they felt that labyrinthitis itself is not an indication to operate immediately. He had had quite a number of cases associated with mastoiditis.

Dr. H. L. Williams believed that all members of the Academy are very grateful to Dr. Friesner for this illuminating pathological discussion, because one cannot understand too clearly the fundamentals of the disease processes. From the paper which Dr. Lillie and Dr. Williams have written in regard to disease of the pyramid, they had had the opportunity of securing complete Roentgenological studies of three cases. It was striking to note the room between the wall of the petrous pyramid and the labyrinth and they thought it feasible to follow the route by which the disease had advanced into the pyramid and at operation this was found to be so. They felt that in these cases of petrous suppuration nothing more would be necessary in most cases than a complete exenteration of the pneumatic cells of the mastoid and the pyramid. A radical mastoid operation can give no better exposure in this type of disease and it should never be necessary, with the possible exception of the presence of peritubal cells which Dr. Friesner has shown are so rarely involved.

Dr. H. L. Rothschild felt it would be difficult adequately to express the delight and complete sense of satisfaction of the members of the Academy with this beautiful presentation of Dr. Friesner. He felt that all were impressed by the brilliance of his laboratory research and the keenness of his observations, and, what impressed Dr. Rothschild most, by the soundness of his deductions.

Dr. Friesner expressed his thanks and appreciation of this honor and his pleasure at coming to address the Academy. He said he would like to leave one thought with the members, that is, go slowly with the case in which the ear is discharging profusely; but immediately go after the case that is not discharging, because the latter is serious. He cited the case of a man who had a pneumococcus III in the mastoid; had no pain, no tenderness and very little discharge from the ear. One ear resolved and then the infection recurred. He was operated and had an extensively diseased mastoid. Along about the seventh week following this operation the man began to complain of pain back of his eye. Dr. Friesner said to his assistant that he was going to operate on the man. That patient made an uneventful recovery; he had a dry ear and a healed mastoid wound. That was two years ago and the man is perfectly well.

BOOK REVIEW.

Emotions and Bodily Changes. H. Flanders Dunbar. A Survey of Literature on Psychosomatic Interrelationships. 1910-1933. New York: Columbia University Press, 2960 Broadway, 1935. Pp. 560. $5.00.

The author presents this survey as an attempt to "bring together in some sort of perspective the research material which has combined to develop those concepts in medicine which are the necessary foundation for further research in the sphere of psychosomatic problems."

Part I is devoted to a discussion of the shift in emphasis in science from the part to the whole and the importance of this viewpoint in problems of acute and chronic illness. A chapter on methodology and measurement stresses the confusion introduced when a correlation to emotion is attempted.

In Part II, the relation of psychical factors and bodily disturbances is considered for the nervous system, musculature, endocrines, general metabolism and heat regulation, the cardiovascular system, the respiratory system, oto-rhino-laryngology, the gastrointestinal system, the genitourinary system, the special sense organs including the skin, and the bones.

Part III deals with therapeutic techniques and pitfalls. The author concludes that the understanding of organismal theory is fundamental to the study of disease processes. Functional and organic can no longer be separated.

Dr. Dunbar has summarized the literature and presented a bibliography that should make this a valuable source book in the field of the interrelationships of emotions and bodily changes. The literature is surveyed, organized and condensed to prove the author's viewpoint that more emphasis should be placed on the psychological side in diagnosis and treatment of diseases and that better training in psychiatry should be an integral part of medicine. H. F. S.